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THE FALLACIES OF SOCIALISM

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This is my first visit to your city of New Orleans, though I have been in your state several times in the last ten years. And it may be of some slight and passing interest to know that this conservative midwesterner has much in common with the people of the south, generally, and New Orleans particularly. For one thing, my heritage is the heritage of the south, for I was born in that part of the Free State of Maryland which found its sympathies and its politics closely identified with the gray of Lee in a glorious, albeit unhappy yesteryear. Having been born on the Eastern Shore of Maryland, which an Eastern Shoreman never allows you to mistake for Baltimore or Western Maryland, I found my roots planted deep and well. I have been happy to grow these past ten years under the rain and sun and wind of the midwest. But this was a nourished cutting and not a transplanting of roots! And as far as New Orleans is concerned, why I knew about your city history almost as soon as I did the history of Baltimore . . . for I went to a McDonogh School. Yes, I have enjoyed the thoughtfulness, the foresight of John McDonogh, too! And as so many of you know, John McDonogh endowed but one school in Maryland . . . outside the city of Baltimore. And in Maryland, the McDonogh School grows in stature and scope with the passing of every year. But twenty-five years ago, when first I entered the school for a most pleasant and tremendously worthwhile four years, the school was small and each of us had lost either our father, or both our parents—for that was as John McDonogh had willed it. And we were boarded at the school and worked on the farm, performing chores during the school semesters, and putting in a good day's work during the summer. This one coincidence may be matched with another, and one that may be appreciated in a small degree by the members of the medical profession. At the time of my entrance into the McDonogh School, the chairman of the board of trustees was Dr. J. M. T. Finney—or Dr. John T. Finney. However you knew him—you knew his reputation. He had by 1924, attained his great prominence in John Hopkins Hospital as an associate of the famed Dr. Halsted and contemporary to Dr. Hugh Young, Dr. William H. Welch, Dr. William Osler, and so many other illustrious names in that hospital's and medicine's hall of fame. Since my father had died in the flu epidemic of the seventeen's and eighteen's, my mother had gone into training as a student nurse in the hospital in Salisbury, Maryland, feeling that she had to be prepared to take care of her sons. After graduation we moved to Baltimore where the possibilities for her nursing career seemed better. Before long she was assigned cases where Dr. Finney met her and learned of her problem and interceded to have me admitted to McDonogh Boarding School, while Dr. Young, with family connections in Vir-

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gina, aided in getting my brother a scholarship at William and Mary. So you see, there is a kindred spirit that I shall keep alive between the McDonogh schools, and will always feel proud of the first association I ever had with the medical profession, and which was tremendously worthwhile. Is it any wonder then—that I now feel so honored that I have chosen to lend my voice to the thinning ranks of the vocal conservative? Is it any wonder that I refuse to take the easy road and coast along downhill with the socialists? The fallacies of socialism are obvious, and yet in this great country of ours too few of its thinkers and builders will stand firm against the tide which even now breaks over us, edging us back, causing moral erosion, and an undertow which pulls at our very fibers. Those who churn the waters in this man-made storm are perpetrating a fraud which has been exposed time after time during the past six thousand years. And yet, in every generation there is a renewed interest in the old theories which begins to capture the imagination of the masses. The self-righteous do-gooders begin to shout as though they have found a new Utopia—while the self-conscious sycophants mumble their approval in an attempt to build a high decibel count to impress the unthinking people. Sadly enough, these people are impressed. For they are many and many people are selfish and many people want something for nothing and if you promise, and *seem* to deliver these unthinking people applaud without reckoning the cost. It's the old shell game, but too many remain at the gambler's table.

There's not much choice between socialism and communism, and in fact, up to 1917 the words were used synonomously—just as today we speak of free enterprise and capitalism synonomously. Karl Marx,—the disgruntled and bilious,—Karl Marx used the words socialism and communism interchangeably in his writings one hundred years ago—and the revolutionists who overthrew the forces which overthrew the Czar named their government a Socialist government, even as Hitler named his government a socialist government. The point to be remembered is that these were dictatorships —predicated upon the ego of a few, but using old clichés and old platitudes to ensnare the unthinking. One can more readily excuse Karl Marx for his fallacious thinking than we can excuse those who today espouse these same theories which time has proved erroneous. The Communist Manifesto and Das Kapital which are the ten commandments and Bible of present day communism, need only be read to inform that the author was laboring under the delusion of a static economy and the belief that working conditions would not be bettered through the machine age which was even then being ushered in by way of the industrial revolution. I do not intend to use the little time I have here in New Orleans to review communism and socialism. Suffice to say that those who oppose socialism are morally and economically right. And that is no play on words. We are right, and if we are right of center in being right, that is fine, too! Let us accept the role of the conservative and emblazon the word on our banners and escutcheon. This discussion may seem to approach a political discussion, but I refuse to accept such a thought as valid unless we admit that most every conversation today is political in varying degrees. Certainly the Glenn Taylor type of Democrat has little in common with the Harry Byrd Democrat. By the same token, the schisms within the Republican party have widened because of this same "socialist-conservative" argument. No matter how the proponents of more governmental control slice it, it comes out the same! The individual is nothing—the strength of the party is the only thing that matters! All means are used to maintain this power, even to liquidating those who were considered a source of strength at one time, but whose differences of opinion become embarrassing. When the central government is given great power, such abortions are easily explained. You need only to look at the record to appreciate these facts. And it is repugnant to me to listen to or to read of some supercilious double-dome attempting to bolster the argument for socialism when history

has recorded the failures of all such forms of governments. Plutarch's account of ancient Sparta is as apt a description of communism as practiced today as though we were to have access to all of Russia for our own report. Let us also appreciate that the Pilgrim Fathers established what could be considered the first commune in America. It started on the Mayflower—and the attempt to carry on and operate Plymouth as a commune continued until they awakened to the stern realities of life. Their theories were good—but they had forgotten that theory and practice do not necessarily follow one after the other. Food and shelter became their prime objective, even as it is yours and my objective today. After half the colony had starved through the first winter's commune, they began to put each person on his own for survival. They learned an important lesson the hard way. After learning, they prospered. Other socialist and communistic settlements were established in the new colonies, but they disappeared as the contrast between the socialist groups and the free enterprisers became too great to be long ignored.

The trouble seems to be that we are all just a little selfish, and when we see that we are not doing too well, we want to know why. We are told certain things about socialism—but the proof is absent. Whenever a communistic community or nation is compared to a nation where there is freedom of enterprise, the contrast is so tremendous as to defy rationalization. That is why the masters of the Kremlin refuse to have a free exchange of news or people. They dare not allow their people to make the contrast. It is much like the tailor who is reluctant to allow you to take the suit to the daylight. You must accept their information and their sales talk as gospel. For years, the sales slogan of the Packard motor car company was—and perhaps it still is—"ask the man who owns one!" And every day we see more and more advertisements offering thirty days free trial. We believe the obvious needs no underlining. We know of no instance where one who has chosen the American way of life has willingly returned to the great planned states. The free enterprise system may seem ruthless to us at times, but to someone who has been under the eyes and heavy hand of the protecting socialist states, our system seems impossibly warm and free. They know—even as we may soon see—that the ruthless tactics necessary to get a socialist state started become increasingly more ruthless in the efforts to conceal the errors and the defects of a scheme that never has been made to work and never will be made to work. Anly the stupid, the selfish, or the fool refuses to accept these facts. The fool accepts the theory that under a socialist government "human equality" and the "brotherhood of man" are recognized. The selfish use these points to create catch phrases and build a following. The stupid simply follow like Mortimer Snerd. All three know that such preachings will be difficult to combat, for we, too, are appreciative of morality. But we also have found that the real nature of man must be recognized. We have learned, they have not—or ignore it.

The planners assume to be acting in the public interest and the welfare of the people. We, they say, must not oppose this, for we are not opposed to the public interest and the welfare of the people, are we? How utterly distasteful you feel to be included among those who have "special interests." And at first the "special interest" groups were few—or so we thought. But many a man who today finds himself attacked as a member of a "special interest" group is half-horrified to recall that only yesterday he was self-righteously pointing a finger at another "special interest" group, and supporting, whether he knew it or not, the cause of the socialists.

Now, this particular "special interest" group which honors me tonight must admit to having a lobby in Washington and must admit to the charge of building a three and a half million dollar fund to fight legislation which it opposes. Yet, in very few instances have the members of the medical profession individually campaigned to keep Mr. Oscar Ewing's program from being

made the law of the land. Are we to suppose that the majority of the members of this profession are *for* socialized medicine? Certainly not! For if that were true, then we have a great number of medical men—thinking people—being led around by the decisions of a few. Our doctors, bless them, are self-conscious about this curious position into which they have been forced.

But each must now make his stand clear. The point to be made in the argument to be presented to the public in these critical days ahead is that the self-interest of the individual citizen is involved. I believe we have pointed out tonight how we may have forgotten that this creeping paralysis was not properly diagnosed soon enough, and that the public generally and the poor people particularly have been living in a fool's paradise of ignorance and promises. It is a simple matter to take a lot and give a little. The tax dollars which will be used to further this collectivist and socialistic plan will greatly affect the incomes of the middle class people and the poor. No one denies that many people not now getting medical attention would be benefitted at first by the services to be rendered. At least, everyone will run for the medical grab bag. These same people, however, may be held personally responsible at times for the lack of medical care. It may seem silly to repeat again that many citizens refuse to avail themselves of present low cost medical care for a number of reasons. Many times because of selfishness. To find people of low income groups filling the taverns and race track apron is simply to observe that the American citizen has heretofore spent his dollar where he wanted to spend it. If the citizen desired to rush his car to the garage mechanic the moment a knock was heard in the motor, that was his business . . . although that same man might tell his wife she's simply getting old when she complained about the stairs. If the situation were reversed would we attempt to legislate to socialize garage mechanics? If we are to legislate that a man be forced to pay taxes in order to be conscientious regarding his own and his family's ailments, we should say so. We must say to the American citizen in forthright fashion . . . and not in the defensive manner of a "special interest" group—that you, Mr. John Q. Public—are already footing a whopping big tax bill—you, Mr. John Q. Public—shell out to the bureaucracy in Washington between 20 and 30 per cent of your earnings every week. You're not getting "something for nothing" in this old world of ours, John! Just look at the recent report of the president's commission to reorganize the government. Examine that report, John, look it over tomorrow night after you and the Missus put the kids to bed and have the dishes cleaned up. Just remember that almost every department in Washington is sloppily run—and expensively run. And just look at the chap who is running around the country telling the people we should have compulsory health insurance because it is in the public interest and that only "special interests" are against it. Ask Oscar Ewing how many of your tax dollars he spent last year for propaganda—and how much he will spend this year. Ask Oscar Ewing if he is a politician with a burning desire for more power or if he is really concerned about the health of the public. Ask Oscar Ewing if this is his idea. Ask Oscar Ewing how many votes does he figure would be available through the use of John Q. Public's tax dollar to build a bigger bureaucracy to administer the socialized health program he desires!

Oscar Ewing is a man, just one of many in the bureaucracy which has been built with tax dollars. He's open to the very same frustrations and desires as any of us. He seems a little frustrated right about now, for he isn't too sure he's going to be able to peddle his little tax-gathering gimmick to the public. And that means that he won't get that Cabinet post to which he aspires. But he'll continue to spend the money to have the propaganda created to try and peddle his Pestle Politic. Not a thought in the world of trying to save his fellow citizens a dollar or two in tax money. No! Oscar Ewing must fight his own special interests: For power and more power. It is obvious now that too many Americans

ignored what was happening to this country through the recent years. The bureaucracy was building, and bureaucracy is always followed by other forms of socialism in order to increase the taxation to pay for the experiments the planners try to sell the people. The doctors and dentists are assumed to be naturally conservative, but we know of few prominent medical man who cautioned us back in 1933 or 1934 — let alone in recent years. Now, suddenly we are faced with the problem of compulsory health insurance and the fingers of the bureaucrats are around our necks and we don't like it. Those who step in the same boat I've been paddling for years might just as well accept the unpleasant fact at once that your arguments and mine are psychologically handicapped by the very nature of man. For we are selling economic morality which in the long run is for the public good. We are competing with economic immorality which looks good and sounds good and offers a short escape from economic reality. For the government cannot give anything until first it takes. And after it has taken it lops off an awfully big slice of the tax dollar before any real return is made to the citizen. Overcoming the psychological handicaps in selling economic morality and conservative thinking in opposition to social fallacies and government taxing and spending can be compared to the handicaps suffered by the wife in opposition to the mistress. The mistress promises glamour, irresponsibility, and escape from the difficult and dull tasks that are essential to decent life. The wife promises very little which is alluring and her appeal must be self-respect, integrity and social responsibility. But the wife is protected by ethics and law, while our thinking has deteriorated to the point where it is fashionable to be sophisticated and sophisticated to accept vulgarities from the mouth of the chief executive in the presence of ladies and gentlemen of Washington's military and social circles. We have noted how this vulgarity had little effect on the public mind. Thus have we been conditioned. For every one person to find it shameful, there were hundreds to hunch an elbow in a friend's rib and say "that's telling 'em off!" Again, evidence that it is easier to sell immorality than morality once the barriers are down. So it is, as we have said, in selling collectivism or socialization as opposed to individualism. Collectivism, socialization, and its ultimate degenerate offspring, fascism or communism appeals to the sadistic instinct of man to destroy those who have more in equipment, resources or talent, appeals to his venal instinct to want something for nothing, appeals to his cowardly instinct to condone evil-doing through mob action, and as we see so clearly today, appeals to his pagan instinct to deny his moral responsibility to God through his conscience. It is impossible to deny that these instincts are universally present in a human being, and we are proving ourselves to be incredibly asinine not to recognize that these instincts have been appealed to since the beginning of time, and certainly during the last several generations by dictators of the left and right. It is easier to be a demagogue than a logician. So it is easier to sell collectivism in any form than to sell individualism. The selfrighteous thinkers, the ignorant and the charlatan, win a ready audience and an immediate following by eager promises which on the surface seem difficult to challenge. But they must be challenged. Not only challenged, but proved to be exactly what they are: The means to an end by power-mad bureaucrats who would, by the sheer weight of politics, be forced to take the next step to full collectivism or socialism and thence to fascism. Let it be noted well that in every plan which has been presented by the collectivists in this country since they sold the administration on the NRA we have had the evilness of the threat of state force. And yet they hide behind the veil of liberalism. But, in every plan there is a desire to take away a personal liberty. And the individual is soon confused into thinking those who would protect his personal liberties are reactionaries while those who would take away these liberties in exchange for a guaranteed security in a brave new world, would

make him like it with state force. Therefore, in fighting the good fight to keep your profession from being controlled by the collectivists you are in honorable combat. Your weapons are words. Your shield is your common sense. You have waited too long for the attack which you should have known was coming. You have waited in the forlorn hope that by some curious reasoning it would stop short of your profession. You ignored the warnings. Now you know. And now you know that the attack will be heavy for the weapons of the enemy have been sharpened and improved through years of application and the arsenal seems unlimited. Yours is an honorable profession. You have made it so. You have a right to defend it.

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CLINICAL USE OF HEPARIN AND DICUMAROL:

METHODS AND PRECAUTIONS

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A. HEPARIN

Jay McLean,[1] working in Howell's laboratory in 1916, discovered an anticoagulant in animal tissues, which Howell[2] later named heparin. Heparin was too toxic for clinical use until purified in crystalline form seventeen years later.[3] It was identified in 1935 by Jorpes[4] as a mucoitin polysulfuric acid, carrying a strong electric charge. It is nature's own anticoagulant, found in the mast cells of Ehrlich, and is now processed chiefly from beef lung tissue. It prevents the clotting of blood by the following mechanisms: (1) It helps to prevent the conversion of prothrombin to thrombin. (2) It forms, with serum albumin, an antithrombin. (3) It prevents the formation of thromboplastin from platelets. Its anticlotting effect is abolished by protamine, as first shown by Chargaff and Olson.[5]

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METHODS

The various methods of giving heparin are outlined in Table I.

Before starting heparin, the clotting time is determined using the Lee-White method modified as described by Loewe,[6] using four clean dry 75 by 10 mm. test tubes. The range of normal clotting by this method is eight to fifteen minutes. The objective is to keep the clotting time above three times the patient's normal (twenty-five to forty-five minutes). If the blood is not well heparinized the coagulation time is determined by failure of the blood to run down the side of the tube during gentle tilting. If the blood is well heparinized, sedimentation of the red cells occurs, and although a plasma fibrin clot may form above, this is disregarded and the moment when the red cell layer ceases to flow when the tube is gently angled is taken as the clotting time. Both physician and technician should study Loewe's meticulous discussion of the care required in performing and interpreting the Lee-White coagulation time (LWCT).

TABLE I.

HEPARIN METHODS

I. *Intermittent I.V.*: Using 10 mg. per cc. Preliminary LWCT 50 mg. heparin I.V. LWCT 10-30 Min. and 4 hours later. If no hyper-reaction continue 50 mg. I.V. every 4 hrs. (one night interval of 6 hrs.) LWCT every 24 hrs. Objective: Maintain LWCT 25-45 min. or at least 3 times the patient's normal LWCT.

II. *Continuous I.V.*: Use only if I.V. fluids required.

III. 200-300 mg. heparin in retarding media S.C. Use ampule without added vasoconstrictors. Clotting time not always adequately prolonged so LWCT needed every 8 hrs. at first. Effective 24 hours.

IV. Concentrated solution 100 mg. per cc. I.M.??

Heparin may be given intravenously by intermittent doses of 50 mg. every four hours except for one interval of six hours during the night. Twenty minutes after the initial dose the LWCT is done to detect hyperreactivity or hyporeactivity; usually at this point it will range between forty-five and seventy-five minutes. At the end of the first four hour period, blood is drawn for

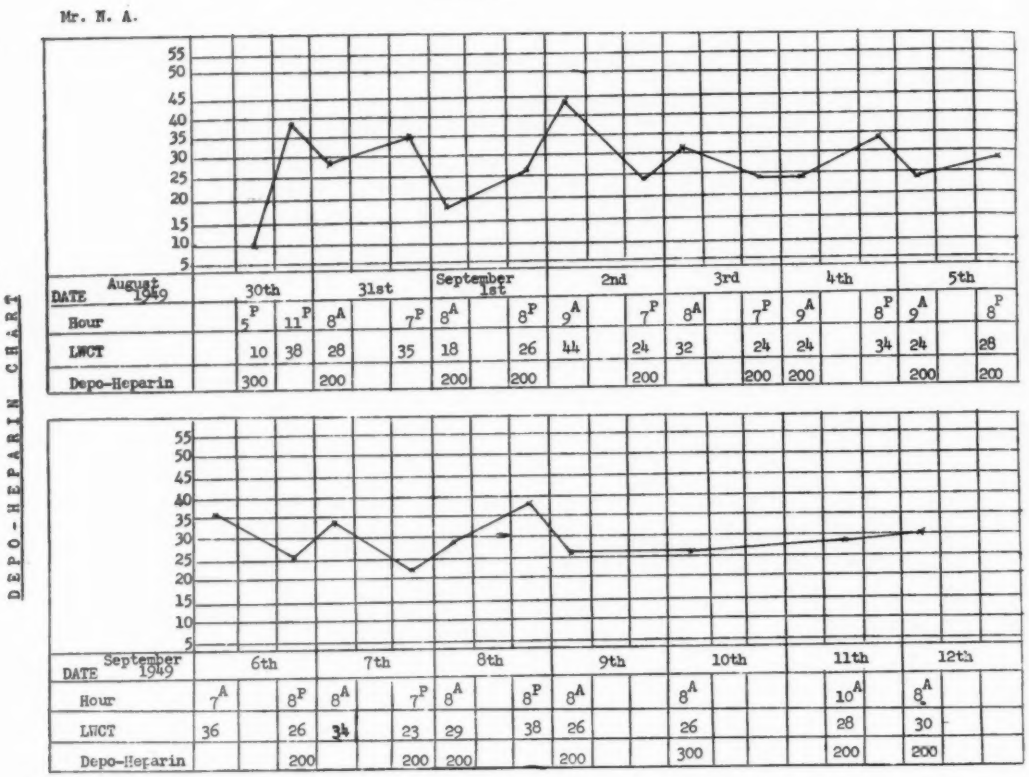
another LWCT and through the same needle the second dose is given. If the LWCT has then returned to normal, as is usually the case, the 50 mg. dose is continued, but if the clotting is delayed to twenty-five minutes or more, the interval may be prolonged to five or even six hours. Thereafter, once during each twenty-four hours, the LWCT is done to avoid excessive heparin effect.

If the patient needs intravenous fluids, 100 mg. of heparin may be added to a 1,000 cc. flask of normal saline or glucose solution, and given at the rate of 40 drops per minute. The LWCT should be taken after half the amount is used to determine sensitivity to heparin, and the rate of flow adjusted accordingly. The effect of heparin given slowly in this fashion wears off abruptly, so no more than two hours should intervene between the intravenous fluids—and if it becomes desirable to omit fluids, heparin should be started immediately by other methods.

The use of heparin in retarding media* is rapidly becoming the method of choice, as the preparations now available are well tolerated. Some long acting preparations are prepared with vasoconstrictors added to further prolong the heparin effect, *but under no circumstances should the ampoule with vasoconstrictors be given any patient with coronary heart disease!* The initial deposit for patients weighing under 150 pounds is 200 mg., and over 150 pounds is 300 mg. At the onset, in arterial occlusions, the patient is "primed" with 50 mg. of regular heparin, intravenously, to avoid the delayed response from the deposit. Every eight hours for the first three to four days the LWCT is checked to avoid underdosage or overdosage. A good rule is to administer 200 mg. whenever the LWCT is found to be less than three times the patient's initial clotting time. After the first few days the interval of testing can be stretched to twelve hours and after seven days to twenty-four hours. By this time the usual pattern of response to a given deposit of heparin has become established so it is permissible to check the coagulation time only on alternate days. Loewe and Eiber[7] advocate using a 400 mg. deposit and state the clotting time will then be effectively delayed for forty-eight hours or more. This has seldom been observed unless a preparation containing vasoconstrictors is used, and the smaller dose at more frequent intervals appears to produce less wide swings in the clotting time. Evans and Boller[8] found the average duration of effect forty-one hours after a 300 mg. deposit without vasoconstrictors, and sixty-one hours when a deposit was used, part of which contained vasoconstrictors. Holden[9] found the clotting time after a deposit of 300-400 mg. of heparin (not stated with or without vasoconstrictors) to be eighteen to fifty-five minutes after twenty-four hours and rarely over twenty minutes at twenty-four hours. Martin, Artz and McCleery[10] found 200 mg. without vasoconstrictors to be effective for twenty-four hours. The term "effective" as used in these reports refers to *any* increase in the coagulation time, not to a coagulation response of three times the normal, which is required for ample protection against thrombosis. The dosage used, date, and hour of the clotting time should be charted in a coagulogram (Figure 1). Heparin may be continued in this way a month or longer but I have encountered no cases in which a single deposit was effective for four or five days as described by Loewe[11] and doubt if heparin will lend itself to long-term cases. After a month, if it seems desirable to continue anticoagulants indefinitely, dicumarol is started, and heparin omitted three days later when the dicumarol will have become effective. In the "switch-over" to dicumarol it must be borne in mind that as long as the LWCT is over twenty minutes the prothrombin time may be prolonged due to the effect of the heparin on the plasma clotting, so the prothrombin time is to be relied on only after the LWCT approaches normal. This renders the "switch-over" following heparin in deposits a little more troublesome than after intravenous therapy, wherein the heparin effect is usually dissipated within three to four hours. It must

*Heparin/Pitkin's Menstruum (W. R. Warner Co.) and Depo-Heparin (Upjohn).

FIGURE I.



also be borne in mind that when dicumarol has become markedly effective as evidenced by prothrombin activity below 20 per cent, there may be some prolongation of the clotting time as well but rarely in the first seventy-two hours of dicumarolization. The study by Long, Hurn, and Barker[12] of the effect of heparin on prothrombin time is informative.

Concentrated *aqueous* heparin intramuscularly is said to induce effective hypocoagulability.[13] The use of caronamide in prolonging the effect of heparin Pitkin menstruum has been described by McCleery, Artz and Sirak.[14]

B. DICUMAROL

Everyone knows the debt we owe Karl Link[15] and co-workers for the discovery and synthesis of dicumarol. In spite of further attempts no better oral anticoagulant has been produced and Link[16] is of the belief that since dicumarol was found in nature it is not likely a less toxic substitute will be synthesized. Although the usual dosage recommended for dicumarol is 300 mgs. the first day and 200 mgs. the second day, it is just as feasible to use 400 mgs. the first day (in divided doses to avoid nausea) and 200 mgs. the second day, and by so doing the height of the dicumarol effect may be reached earlier. In order to avoid overdosage in patients who are sensitive to dicumarol, the dose on the third day should be omitted, and thereafter the daily dose is variable dependent upon the prothrombin time (Table II). The use of divided doses of dicumarol may produce less fluctuation in the prothrombin time,[17] thus if the patient requires 100 mgs. daily, 25 mgs. four time a day, or 25 mgs. morning and night is better than 50 mgs. once daily. In some patients, even long term cases, it is not always easy to adjust the dosage of dicumarol so that a satisfactory prothrombin time bracket is maintained, and this is particularly true for the novitiate. The daily re-

quirement varies from 25 to 150 mgs.; the usual requirement is 75 mgs. It is important that a chart is kept showing in graphic form the daily prothrombin time, preferably both of whole plasma and 12.5 per cent dilution, and dosage of dicumarol. Visualizing the trend on the chart is of great help in following the anticoagulant effect.

TABLE II.

DICUMAROL DOSAGE PLAN BEGINNING 4th DAY

Prothrombin Time	Dosage Dicumarol
Over 35 seconds	No dicumarol
30-35 seconds	25 mg. only
25-30 seconds	25 mg. b.i.d.
20-25 seconds	25 mg. t.i.d.
Under 20 seconds	50 mg. t.i.d.

Above schedule applies only if laboratory uses a thromboplastin which by Quick or Link-Shapiro method gives normal prothrombin time of 11-14 seconds.

Quick's one-stage method of prothrombin determination and the Link-Shapiro modification of this method, are now generally used. Quick's method utilizes rabbit brain thromboplastin* and the Link-Shapiro modification utilizes rabbit lung thromboplastin**. The Link-Shapiro test calls for determination of the prothrombin time in 12.5 per cent saline dilution of plasma, as well as in whole plasma, which is valuable in detecting thrombotic tendencies and also serves to warn of possible hemorrhage. The results of the prothrombin time determination are expressed in seconds. Anyone who has followed the arguments in the current literature must be aware of the disparity of opinion as to what constitutes adequate laboratory control of dicumarol therapy. For several years it has been argued that in order to accurately gauge dicumarol therapy it is necessary to report the test in terms of percentage of prothrombin activity, which necessitates interpolation of the results on a logarithmic curve constructed from average prothrombin times of serial dilutions of a number of normal plasmas. But errors may enter into the construction of such a hyperbolic curve, and in view of the fact that pathologic plasmas or dicumarol treated plasmas cannot be compared precisely with normal plasmas in serial dilutions, regardless of whether normal saline solution or prothrombin free plasma is used as a diluent, the value of transposing the prothrombin time in seconds to percentage of prothrombin activity is greatly vitiated. Furthermore, reduction of the prothrombin activity from 100 per cent to 30 per cent is associated with so few seconds increase in prothrombin time of whole plasma that the difference is practically within the limits of accuracy of the test. Therefore, reports in terms of prothrombin activity by a laboratory using the one-stage method of Quick or the Link-Shapiro modification, have no practical significance unless the the prothrombin activity is below 30 per cent. Shapiro and Weiner[18] state: "It seems to us that to follow adequately the prothrombin response to 'dicumarol', the clinician should know the normal range of the thromboplastin used and the therapeutic range he wishes to establish in terms of time. With this knowledge the calculation of percentage is superfluous; without it the percentage figure is misleading. Of additional importance is the fact that use of figures as percentage of normal may rapidly acquire the connotation of a sense of accuracy which has not been proved. Estimation of prothrombin times is, at best, an indication of the in vitro clotting capacity of a specimen of plasma obtained and studied under certain standardized conditions."

Only time and more studies will incontrovertably settle the question of the proper level of prothrombin deficiency to set one's sights for in dicumarol therapy. I am inclined to agree with Barker and associates[19] who, in December 1947, stated: "When the concentration of prothrombin was kept between 10 and 30 per cent of normal few instances of major bleeding and almost no instances of thrombosis have occurred. This zone of effectiveness and relative safety is a comparatively narrow one and some observers have been satisfied with less drastic reduction in the concentration of prothrombin. This may be sufficient to prevent thrombosis in some patients but is certainly

*A reliable commercial product is supplied by Difco Company.

**A reliable commercial product is supplied by Maltine Company.

less effective if the stimulus to thrombus formation is strong. *We believe that if dicumarol is used at all, a strong attempt should be made to secure the optimal effect in each patient.*"

A practical rule is to maintain the prothrombin time between two and two and a half times the average normal expressed in seconds obtained by identical technic using the same thromboplastin, in which event the percentage of prothrombin activity will invariably fall between 30 per cent and 10 per cent, a safe yet effective bracket. Several normal plasmas should be tested each day as the commercial thromboplastins vary slightly from batch to batch in potency.

The efficacy of dicumarol in preventing intravascular thrombosis has been questioned because occasionally intravascular thrombosis has occurred in spite of apparently adequate dicumarol dosage. Such instances are often not attributable to failure of dicumarol but rather to failure of the laboratory to render accurate prothrombin tests or to failure of the clinician to anticipate the dicumarol dosage adequately. The disadvantages of both dicumarol and heparin are outlined in Table VI. The delay in obtaining a therapeutic depression of prothrombin activity is obviated by the temporary use of heparin.

Although no set dosage can be established, and each case must be adjusted individually, yet the schedule outlined in Table II can be used as a guide usually, provided the technic and the thromboplastin used gives a prothrombin time of eleven to fourteen seconds in whole plasma of normals.

Since dicumarol is excreted by the kidney any degree of abnormal urea retention was formerly considered a contraindication to the use of dicumarol, but in my opinion only severe renal impairment constitutes a definite contraindication, while in cases showing moderate urea retention the use of smaller doses than commonly used is safe and effective provided extra care is exerted. The ordinary dose may be given in the presence of albuminuria and casts without urea retention, unless the abnormal urinary findings are due to heart failure, in which case since hepatic congestion is often associated, smaller doses are indicated.

Jubelirer and Glueck[20] recently studied capillary fragility studies in 100 patients receiving dicumarol and found no correlation between the Gothlin index and occurrence of hemorrhage from dicumarol. Dicumarol inhibits intravascular thrombosis, but in glass tubes the coagulation time is not prolonged significantly until the prothrombin activity is less than 10 per cent of normal, and this is not a constant finding. However, in silicone treated tubes the coagulation time for several hours up to forty-eight hours is prolonged when the prothrombin activity is depressed to 20 per cent or less of normal. Moloney[21] and coworkers first showed this, and I have confirmed their observation many times in routine dicumarol treated ambulatory subjects. However, the care required in performing coagulation studies in silicone treated tubes is more bother than performing a one-stage prothrombin time, so the procedure will not replace the prothrombin test and furthermore the duration of hypocoagulable effect from only one hour to twelve or twenty-four hours is subject to such great variability that such coagulation data cannot be used as a guide in therapy. Knisely's[22] recent work on blood sludging in relationship to disease and the clotting mechanism, lead to the observations of Laufman, Martin and Tanturi[23] that heparin and dicumarol do not prevent sludging but prevent the sludged masses of cells in the capillaries from adhering to the endothelial lining of the vessel, thereby preventing thrombus formation in the presence of sludge.

Although a spectro-photometric method for the estimation of dicumarol in plasma or urine has recently been devised by Axelrod, Cooper and Brodie[24] it is not likely that this will replace prothrombin determinations in guiding dicumarol usage, though it should add to our knowledge of the pharmacology of the drug.

HEMORRHAGE FOLLOWING THE USE OF ANTICOAGULANTS

One per cent protamine sulfate has been shown to be a clinically effective and safe antidote[25] for heparin. The amount of protamine sulfate used should be calculated approximately milligram for milligram based upon the amount of heparin which it is desired to counteract.

Following the use of dicumarol, bleeding is due to excessive hypoprothrombinemia. Vitamin K either in the form of K-1 oxide[26] or synthetic vitamin K,* Menadione bisulfite,[27] often reduces the bleeding tendency if given intravenously in adequate dosage. Since gross hematuria will usually clear up on withdrawal of dicumarol within a few days, it may not be wise to use vitamin K intravenously for this complication because of the possibility of increasing the tendency for clots to form within the kidney producing subsequent ureteral colic, a distressing aftermath I have witnessed three times. Transfusion of blood or plasma is of value in brisk hemorrhage whether due to dicumarol or heparin, aiding the restoration of prothrombin deficiency as well as offsetting the heparin effect and in addition replacing the needed blood or plasma volume. The use of ascorbic acid and rutin may serve to avoid hemorrhage.

The incidence of hemorrhage following the use of anticoagulants depends to some extent on the philosophy of the clinician, who may be inclined to "push" the dosage in the belief that it is more desirable to assure patients with cardiovascular disease full protection against thrombo-embolic complications, thus running some risk of hemorrhage, rather than to use a dose carrying with it little chance of hemorrhage that might fail to inhibit intravascular clotting. In using dicumarol, hemorrhage rarely occurs in any event until the prothrombin activity is depressed to 5 per cent unless some pathologic lesion is present in the kidney, liver, gastrointestinal tract, lungs, or brain. Loewe and Eiber[7] said in comparing heparin and dicumarol therapy in coronary artery thrombosis, "It is comforting to know that injection of larger doses to insure satisfactory heparinization does not invite the hazard of excessive doses of dicumarol. In the patient with an intact cardiovascular apparatus there is little or no risk of hemorrhage, even following excessive amount of heparin sufficient to elevate the coagulation time considerably beyond the requisite level." I cannot fully agree, as concomitant ulceration of any serous membrane caused by any pathological process may lead to hemorrhage when either heparin or dicumarol is used, but admit there is decidedly less purpura and hematuria with heparin than dicumarol, not only because heparin is a natural anticoagulant but probably because the heparin effect wears off more rapidly and the laboratory control is simpler.

Hemorrhage following the use of anticoagulants is usually classed as minor or major, the latter including hemoptysis, gross hematuria, severe ecchymosis or generalized purpura, tarry stools, hematemesis, clinical signs of cerebral or subarachnoid hemorrhage, or any bleeding in the central nervous system, heart, lungs, or abdominal viscera, found at autopsy.

The final report of the American Heart Association Committee[28] will reveal the incidence of minor and major bleeding in 442 "control" cases as 5.9 per cent, and in 589 "treated" cases 9.2 per cent incidence of hemorrhage probably due to anticoagulants, and 6.1 per cent probably due to other causes.

TABLE III.

INCIDENCE OF MAJOR HEMORRHAGE FOLLOWING DICUMAROL REPORTED IN SERIES OF CONSIDERABLE SIZE

Author	Year	Cases	Major Hemorrhage
Bruzelius	1943	1,645	38
Allen-Barker et al.	1947	2,307	43
Smith-Mulligan	1948	2,353	26
Wise-Loker-Brambel	1949	3,304	79*
		9,609	186 = 1.96%

*Includes moderate and minor hemorrhage also.

Table III indicates the incidence of major hemorrhage encountered in several large series but does not include all clinical re-

*The 72 mg. ampule of Abbott's Hykinone yields 60 mg. of menadione bisulfite.

ports. A questionnaire was sent out by the author last fall relative to the incidence of hemorrhage from both heparin and dicumarol and the data obtained will be published.[29] In approximately 15,500 treated cases, most of whom received only dicumarol, the incidence of major hemorrhage reported was 2 per cent. The chief sites were in the urologic tract, operative wound, gastrointestinal tract, rectum, lungs, and skin. The incidence of hemorrhage in my experience has been somewhat higher due in part to my belief in the necessity of insuring full protection against thromboembolism. In 160 patients treated for acute myocardial infarction, and in 81 other cardiovascular cases, the incidence of major hemorrhage was 10 per cent. In 54 long-term ambulatory patients given dicumarol for months and years in an attempt to prevent attacks of coronary thrombosis the incidence of hemorrhage was 20 per cent. The details of the hemorrhagic episodes in these cases are discussed in full elsewhere.[29]

Recently Duff and Shull[30] reviewed the literature and reported 23 deaths, including 2 cases of their own, attributable to the use of dicumarol. In addition there have been 5 deaths recorded in the literature following heparin, 4 deaths with dicumarol in my own experience described in detail elsewhere, and 3 similar unreported deaths have come to my attention. Among the 15,500 patients reported upon in the questionnaire, 28 or .18 per cent, died apparently as a result of hemorhage induced by anticoagulants. Thus a total of 63 cases are now known to have died with hemorrhage following dicumarol or heparin.

TABLE IV.

HEMORRHAGE WITH DICUMAROL THERAPY

Rarely occurs until prothrombin activity drops to 10% or even 5% unless pathologic lesion present in kidney, gastrointestinal tract, lungs, or brain.

Hemorrhage controlled usually 72 mg. vitamin K, I.V. Repeated in few hours and 6 hours later. Vitamin K-1 oxide more effective.

Transfusions seldom needed except for replacement.

Bruising and mild hematuria less if rutin used?

Lumbar puncture, lumbar procaine block, or thoracentesis may induce hemorrhage.

Table IV is a brief summary relating to dicumarol induced hemorrhage. Certain precautions should be heeded as a result of past experience. One is the danger of performing surgery or even lumbar procaine blocks when an anticoagulant is in effect. Thoracentesis, paracentesis, lumbar puncture, and spinal anesthesia, should be avoided for fear of inducing hemorrhage from the serous membranes. In a case of acute myocardial infarction not clearly diagnosed, the possibility of the presence of dissecting aortic thoracic aneurysm should be ruled out, as this error in diagnosis was followed by death from hemorrhage in 2 instances. Clinicians should acquaint themselves with the laboratory methods of prothrombin determination and Lee-White coagulation time, and should make it their business to see that the laboratory performs the technic meticulously, or else refrain from the use of anticoagulants altogether.

TABLE V.

ANTICOAGULANT THERAPY

INDICATIONS

After:

Vascular surgery.
Pelvic operations.
Abdominal operations, especially if history of thrombo-embolism.
Splenectomy.
Parturition.

During:

Thrombophlebitis or phlebothrombosis.
Pulmonary embolism or infarction.
Arterial thrombosis or embolism.
Acute myocardial infarction.
Cerebral thrombosis or embolism.
Mesenteric thrombosis or embolism.
Retinal thrombosis, arterial or venous.
Pneumonia, protracted in spite of antibiotics.
Toxemia of pregnancy.
Convalescence after hip fractures.

Long-term:

Congestive heart failure, even without embolism.
Thrombo-embolism due to auricular fibrillation.
After recovery from acute myocardial infarction.
Selected cases of retinal thrombosis.

Recurrent thrombophlebitis and persistent phlebitis migrans.

CONTRAINDICATIONS

Severe liver disease.
Hemorrhagic diathesis.
Subacute bacterial endocarditis.
Recent operations on central nervous system.
Immediately after lumbar procaine blocks or spinal anesthesia.
In advanced renal disease: Use heparin, not dicumarol.
In moderate renal or liver disease: Heparin may be used or dicumarol may be used in small doses.

SUMMARY

Table V summarizes the indications and contraindications for anticoagulant therapy, and Table VI summarizes the disadvantages of dicumarol and heparin.

TABLE VI.

DISADVANTAGES OF DICUMAROL

No standard dose. Unpredictable response. Laboratory technic should be supervised by interested clinician unless numerous tests are being run including several normals daily.

Possible effect on blood clotting factors other than prothrombin formation in the liver.

Minimum prothrombin depression necessary to prevent thrombo-embolization varies.

Anticoagulant effect 1st 48 hours inadequate.

DISADVANTAGES OF HEPARIN

Intravenous adminstration irksome or difficult.

Unpredictable effect of heparin/Pitkin menstruum.

Methods of the use of both heparin and dicumarol have been outlined, and the incidence of hemorrhage and deaths from hemorrhage has been emphasized. Once again the strict necessity of meticulous care in the use of anticoagulants has been pointed out.

REFERENCES

1. McLean, J.: The thromboplastic action of cephalin. Am. J. Physiol., 41:250, 1916.
2. Howell, W. H.: Harvey Lectures, 12:272, 1916-1917.
3. Charles, A. F. and Scott, D. A.: Studies on heparin. J. Biol. Chem., 102:425, 1933.
4. Jorpes, J. E.: On the chemistry of heparin. Biochem. J., 29:1817, 1935.
5. Chargaff, E. and Olsen, K. B.: The influence of protamine on the anticoagulant effect in vivo. J. Biol. Chem., 122:153, 1938.
6. Loewe, L.: Anticoagulation therapy with heparin/Pitkin menstruum in thrombo-embolic disease. Am. J. Med., 3:447, 1947.
7. Loewe, L. and Eiber, H. B.: Anticoagulation therapy with heparin/Pitkin menstruum in the management of coronary artery thrombosis and its complications. Am. Heart J. 37:701, 1949.
8. Evans, J. A. and Boller, R. J.: The subcutaneous use of heparin in anticoagulation therapy. J. A. M. A., 131: 879, 1946.
9. Holden, W. D.: Treatment of deep venous thrombosis with reference to subcutaneous injection of heparin and use of dicumarol. Arch. Surg., 54:183, 1947.
10. Martin, M., Artz, C. and McCleery, R. S.: Some recent advances in the use of anticoagulants with special emphasis on heparin in retarding media. The Health Center J., Ohio Sate Univ., 1:119, 1948.
11. Loewe, L.: Personal communication.
12. Long, M., Hurn, M. and Barker, N. W.: Effect of heparin on the prothrombin time. Mayo Clinic Bull., 21: 225, 1946.
13. Stats, D., and Neuhof, H.: Concentrated aqueous heparin: A new form of intramuscular administration. Am. J. M. Sc., 214:159, 1947.
14. McCleery, R. S., Artz, C. P. and Sirak, H. D.: Practical application of the heparin-caronamide reaction to the potentiation of heparin in gelatin menstruum. Surg., 24:348, 1948.
15. Link, K. P.: The anticoagulant from spoiled sweet clover hay. Harvey Lectures, 39:162, 1943-1944.
16. Link, K. P.: Can we look forward to better anticoagulants that heparin and dicumarol? Chicago Med. Soc. Bull., March 26, 1949, p. 748.
17. Fassett, D. W. and Nichol, E. S.: Absorption and potency of small divided doses of dicumarol in man. Federation Proc., 7:1, 1948.
18. Shapiro, S., and Weiner, M.: Estimation of prothrombin time. Correspondence J. A. M. A., 136:418, 1948.
19. Barker, N. W., Hines, E. A., Jr., Kvale ,W .F. and Allen, E. V.: Dicumarol: Its action, clinical use and effectiveness as an anticoagulant. Am. J. Med., 3:634, 1947.
20. Jubelirer, R. A. and Glueck, H. I.: Capillary fragility studies (Gothlin test) on one hundred patients receiving dicumarol. J. Lab. & Clin. Med., 34:448, 1949.
21. Moloney, W. C., Murphy, A. S. and Harrington, W. J.: Prolongation of the coagulation of whole blood by dicumarol in man. Am. J. Med., 5:40, 1948.
22. Knisely, M. H., Block, E. H., Eliot, T. S. and Warner, Louise: Sludged blood. Science, 106:431, 1947.
23. Laufman, H., Martin, W. B. and Tanturi, C.: Effect of heparin and dicumarol on sludge formation. Science, 108:283, 1948.
24. Axelrod, J., Cooper, J. R. and Brodie, B. B.: Estimation of dicumarol, 3,3'-Methylenebis (4-Hydroxycoumarin) in biological fluids. Proc. Soc. Exper. Biol. and Med., 70:693, 1949.
25. a. Cowley, L. L. and Lam, C. R.: The neutralization of heparin by protamine. Surg. 24:97, 1948.
    b. Parkin, T. W. and Kvale, W. F.: Neutralization of the anticoagulant effects of heparin with protamine (Salmine). Am. Heart J., 37:333, 1949.
26. a. Davidson, C. S. and MacDonald, H.: The effect of vitamin K-1 oxide on hypoprothrombinemia induced by dicumarol. New England J. Med., 229:353, 1943.
    b. Lucia, S. P. and Aggeler, P. M.: Treatment of dicumarol induced hypoprothrombinemia hemorrhage with vitamin K-1 oxide. Proc. Soc. Exper. Biol. & Med., 56:36, 1944.
    c. James, D. F., Bennett, I. L. Jr., Scheinberg, P. and Butler, J. J.: Studies on dicumarol in human beings: Its neutralization by vitamin K-1 oxide, menadione bisulfite, synkavite and blood. Abstracted in Am. J. Med., 1:445, 1948.
27. Cromer, H. E. and Barker, N. W.: The effect of large doses of menadione bisulfite (synthetic vitamin K) on excessive hypoprothrombinemia induced by dicumarol. Proc. Mayo Clinic Bull., 19:217, 1944.
28. Wright, I. S., Marple, C. D. and Beck, D. H.: Report of the committee for the evaluation of anticoagulants in the treatment of coronary thrombosis with myocardial

infarction. Am. Heart J. 36:801, 1948. (Augmented data to be published in final report.)

29. Nichol, E. S.: The risk of hemorrhage in anticoagulant therapy. (To be published.)

30. Duff, I. F. and Shull, W. H.: Fatal hemorrhage in dicumarol poisoning. J. A. M. A., 139:762, 1949.

DISCUSSION

(3) *Question*:

In my practice it is not possible to obtain reliable determination of prothrombin time daily on patients who might need dicumarol. Would it be reasonably safe without determination of prothrombin time to give a patient dicumarol for three to four days or longer, provided there was no clinical evidence of liver disease or kidney disease? If this would be reasonably safe, would it probably be effective in the prevention of thrombosis or embolism?

Answer: Dr. E. Sterling Nicol:

I am sure if you heard the previous discussion you would know the answer. No doctor has the right to give a patient dicumarol unless he has available a laboratory that can furnish good prothrombin determination. We have seen patients on consultation in different communities that have been given, for example, 50 milligrams a day and were told to take it because that much would be safe, yet hemorrhagic phenomena developed. It is not safe to give any amount; even 25 milligrams a day wtihout laboratory control! You should give an adequate dose as many physicians have undertreated patients and have been disappointed. Please do not ever give any patient dicumarol unless you have prothrombin time run. The first week or ten days you start using dicumarol you need a daily prothrombin time. After a week or ten days, ordinarily you can "stretch" the interval of testing to every other day. By the third week you can ordinarily increase the test interval to every third day. You can save your patient one venepuncture if you want to on the second day as there is not much point in checking the time because there will be no effect from dicumarol that early. Make the test at the start, then wait forty-eight hours.

(4) *Question*:

As I understand it, heparin is the only anticoagulant that most general practitioners can use with safety. What would be the best method for a general practitioner to use this agent?

Answer: Dr. E. Sterling Nichol:

For the man in general practice, if the patient is in the hospital, it can be given every four hours. It just happens that the hospital where I do work has "intravenous nurses" who work on eight hour shifts and we get good intravenous work done "around the clock". (I would rather use these nurses than interns, who are not always expert at intravenous therapy.) If the patient is not hospitalized, give heparin in Pitkin menstruum, 200-300 mgs. without vasocontrictors, and then test the Lee-White clotting time every twelve hours. Keep the clotting time at least three times normal, that is, twenty-five to forty-five minutes, and repeat the dose whenever the clotting time is less than three times normal. The physician can check the clotting time if a laboratory is not available, using the technic employing a cc. of blood in each of four small glass tubes. Do not tilt the first tube until five minutes; the second after another five minutes has past, and the same way with the third tube. Tilting the tube too often alters the clotting time.

As a matter of fact, in our practice my associates and I carry such tubes in our bags and when we see a patient getting heparin we run a clotting time at the bedside. Heparin in deposits with long acting media once a day in the average case will work fairly well, but if the clotting time is under twenty minutes, a second daily injection of 100 mgs. will usually suffice. Dr. Leo Loewe says in his experience deposits of 400 mgs. of heparin last forty-eight hours. Dr. Loewe has used more with vasoconstrictors than I have used in coronary cases and he probably gets satisfactory prolongation of clotting for forty-eight hours. Without vasoconstrictors 300 milligrams is usually effective not more than twenty-four or thirty-six hours. Give it deep subcutaneously, not intramuscularly, laterally in the thigh; not in the buttocks where the patient will lie on it. Do not apply cold to the site of injection unless you want to delay effect of the heparin. Do not apply heat or massage unless you want to speed it up. I think this form of heparin will probably prove to be a satisfactory type of treatment. It is less painful than it used to be, particularly depo-heparin.

(8) *Question*:

In extreme old age, such as you run into—eighty-eight and ninety year old patients with fractured hips—is there a contraindication for dicumarol or heparin?

Answer: Dr. E. Sterling Nichol:

I do not believe; it is because in some people that age the vascular status is better than mine may be. One paper was published recently cautioning against the use of dicumarol in patients with hypertension. I do have a little feeling that maybe in the older age group, with marked hypertension, we may run more risk of cerebral hemorrhage. We have had 2 cases of cerebral bleeding at autopsy, 1 microscopic, 1 gross, due to rupture of the lenticulo-striate artery. As far as the age factor itself, I do not think that interferes with the use of it. Dr. Irving Wright, Dr. Nelson Barker, and other experts, feel as I do about age not being a limiting factor.

THYROIDITIS*

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It is the authors' opinion that both acute and chronic thyroiditis are more common in certain sections of the United States than in others. In the Gulf States goitre is not common as compared to the Great Lakes region. The incidence of chronic thyroiditis in comparison to the relatively small number of goitres is higher than the reported incidence from other sections of this country.

ACUTE THYROIDITIS

There has been little written concerning acute thyroiditis, and figures relative to the incidence of this condition throughout the country are not obtainable at present. This is due to the fact that these patients do not undergo operation as a rule and no pathological material is available for study.

Within the past ten months the authors have treated 4 patients with acute thyroiditis. All of these cases occurred in married women. The age incidence varied from the youngest of 33 years of age to the oldest of 59 years of age. All 4 complained of a pressure feeling in the neck, tenderness on palpation of the thyroid gland and the presence of swelling in the neck. Weakness and fatigue were prominent symptoms in 2 of the patients and nervousness was a prominent symptom in 2. One of these patients had fever of two weeks' duration and she had obtained some temporary relief with injections of penicillin. Three of the patients had bilateral enlargement of the thyroid and the fourth had enlargement of the right lobe only. None of this group had an elevated basal metabolic rate.

On examination of an individual with acute thyroiditis the gland is found to be firm on palpation and quite tender. This latter symptom was so marked in one patient she was unable to wear any type of close fitting collar.

The treatment of acute thyroiditis is conservative. Surgery should be avoided. The inflammatory process will clear up promptly as a rule. Lugol's solution and propylthiouracil are of doubtful value. Lugol's solution may be tried and at times seems to be of benefit. In the presence of an elevated basal metabolic rate, propylthiouracil in small doses may be given, but the patient must be observed frequently, as myxedema may ensue. One of our patients in this group was treated with propylthiouracil for a period of four weeks and developed a moderately severe myxedema which responded to thyroid extract. The application of heat or cold to the neck affords symptomatic relief at times.

There is little or no evidence in the literature to indicate that acute thyroiditis is a forerunner of chronic thyroiditis.

INCIDENCE OF CHRONIC THYROIDITIS

For this study of chronic thyroiditis a series of 172 consecutive thyroidectomies from the Mahorner Surgical Services at the Southern Baptist Hospital and the Touro Infirmary in New Orleans was studied. There was a total of 7 cases of chronic thyroiditis in this series, an incidence of 4.06 per cent. Cases of chronic thyroiditis associated with hyperplastic or adenomatous goitre were not included in this group.

In this series of 7 cases of chronic thyroiditis there were 4 cases of Hashimoto's disease or struma lymphomatosa, an incidence of 2.32 per cent; 2 cases of Riedel's struma, an incidence of 1.16 per cent, and 1 case of chronic nonspecific thyroiditis, an incidence of .58 per cent.

McSwain and Moore,[1] in a review of the literature in 1943, found the incidence of struma lymphomatosa to be less than 1 per cent. Marshall *et al*,[2] in a series of 25,000 thyroidectomies, found an incidence of .75 per cent of chronic thyroiditis. Boyden

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et al,[3] in 1935, reported an incidence of .36 per cent of Riedel's struma in a series of 2500 consecutive thyroidectomies from the University of Michigan. DeCourcy[4] reported an incidence of 2 per cent of Riedel's struma in his study of thyroidectomies in Cincinnati.

A review of the record room statistics of the Touro Infirmary in New Orleans reveals an incidence of 8.0 per cent thyroiditis during the ten year period of 1938 through 1948. A review of the record room statistics of the Southern Baptist Hospital in New Orleans reveals an incidence of 8.6 per cent of thyroiditis during the twenty year period 1926 through 1946. These figures were taken from the record room statistics of the two hospitals and were therefore open to criticism, as no review was made of the individual clinical records.

Therefore, since these percentages seemed unusually high, a study of all the thyroid specimens removed surgically at the Southern Baptist Hospital during the period January 1, 1945, to April 1, 1949, was undertaken. The total number of surgical thyroid specimens was 222. The slides in all cases diagnosed as chronic thyroiditis during this period were reviewed and only those cases retained that could be classified as a primary disease. The number of cases thus diagnosed as chronic thyroiditis was 18 out of the total of 222 thyroidectomies, or 8.11 per cent.

This group of 18 cases was then subdivided into three classifications. There were 9 cases of struma lymphomatosa, an incidence of 4.05 per cent; 3 cases of Riedel's struma, an incidence of 1.35 per cent, and 6 cases of chronic nonspecific thyroiditis, an incidence of 2.70 per cent.

It is probable that the diagnosis of chronic thyroiditis is at times used as a discharge diagnosis with a pathology report of hyperplastic or adenomatous goitre showing marked inflammatory changes. These cases should not be classified as a primary chronic thyroiditis and all such cases have been eliminated from our study.

The review of the slides at the Southern Baptist Hospital indicates that the record room statistics at these two hospitals are fairly accurate and an incidence of 8 per cent or slightly higher of chronic thyroiditis can be accepted. This figure is considerably higher than the incidence of 4.06 per cent of chronic thyroiditis in our series of 172 thyroidectomies. The former percentage may be more accurate as it represents a larger number of cases over a longer period of time.

SEX

Riedel's struma may occur in both male and female, but is more common in women. Hashimoto's disease in men is rare and occurs almost exclusively in women. Chronic nonspecific thyroiditis may occur in males but is more common in women. All 7 of our cases of chronic thyroiditis occurred in women.

PATHOLOGY

Chronic thyroiditis, excluding the rare specific types, such as luetic or tuberculous, may for all practical purposes be divided into three groups:

1. Struma lymphomatosa or Hashimoto's disease
2. Riedel's struma
3. Chronic nonspecific thyroiditis.

Group 3 includes those cases of chronic thyroiditis that do not fit in group 1 or 2 and that are determined to be a primary thyroiditis rather than thyroiditis associated with hyperplastic or adenomatous goitre.

A study of the literature on this subject indicates that the giant cell type of DeQuervain should be included in group 3. The giant cell type of DeQuervain, according to Schilling,[5] is one variant of the protean manifestations of the thyroid gland to inflammation of bacterial origin.

In Hashimoto's disease, the thyroid gland shows grossly a diffuse regular enlargement. The surface shows a fine nodularity which is so marked at times that the preoperative diagnosis of adenomatous goitre is made. The capsule of the gland remains intact and with the exception of the pretracheal region is free of adhesions. The tissue is rubbery in consistency and is generally pale white in color, but occasionally

may exhibit a brown-yellow cast. The cut surface of the gland appears divided into irregular lobules by fine fibrous trabeculae. There is present no normal thyroid tissue, little degeneration, and no necrosis or hemorrhage. As this condition occurs almost entirely in women, Schilling[5] called attention to the possibility that the functional demands on the thyroid in women may be a factor in the pathogenesis of struma lymphomatosa.

Microscopically, there is diffuse involvement of the entire gland which consists of a diffuse infiltration of lymphocytes. Numerous lymph follicles with germinal centers are present. The acini are small with oval to slitlike lumens. Generally, the epithelium of the acini is of the high cuboidal type with little or no evidence of degeneration. Occasionally, the lumen of an acinus is filled with a homogeneous mass which contains what appears to be degenerating nuclei. However, larger acini containing colloid are present which contain low cuboidal epithelium showing various stages of degeneration. Numerous large pale polygonal cells suggestive of the Hurthle type may be found arranged in acini, chords, and sheets. The cytoplasm of these cells usually takes an intense eosin stain. The nuclei of these cells vary in size and shape, are eccentrically placed, and contain fine chromatin and one or two small nucleoli. Fibrosis is present as a delicate interlacing structure and does not assume the dense sclerotic appearance that is associated with Riedel's struma. The blood vessels rarely show the changes associated with the latter condition.

In Riedel's struma, the gross appearance varies with the age of the process. The thyroid gland often presents a unilateral involvement. In the early stage, the gland is increased in size, is white in appearance, and is hard in consistency. At this stage the pericapsular adhesions may be minimal. The cut section of the involved gland is semitranslucent, pale white in color. DeCourcy[4] believes the disease process begins as a perithyroiditis rather than as an extension outward from the gland.

In the late stage, the gland is decreased in size, is grayish white, and is bonelike in its hardness. The pericapsular adhesions are diffuse and dense. The cut surface of the involved gland is opaque grayish white and contains an occasional semitranslucent area.

The microscopic picture in Riedel's struma also varies with the age of the disease. Fibrosis is the conspicuous element in all phases. In the early stage there is an acute degeneration of the acini in the involved area with the acini in the uninvolved regions being unaffected. The colloid seen in the involved areas is usually intercellular and contains nuclei of the degenerated epithelial cells producing the psuedogiant cells of Riedel's struma. The fibrous tissue is arranged in whorls surrounding and sometimes replacing the areas of degenerated acini and psuedogiant cells, giving an appearance of a granulomatous lesion. There is a dense infiltration of lymphocytes and leukocytes. Plasma cells, histiocytes, and true giant cells are also present. Occasionally there is a periarteriolar fibrosis and intimal thickening of the blood vessels in the involved areas.

In the late stages, the acini in the involved areas are markedly compressed or entirely absent with the acini in the uninvolved tissue being unaffected. Fibrosis is dense and sclerotic with hyalinization in some areas. A moderate number of lymphocytes and leukocytes may be present between the fibrous strands. An occasional lymph follicle with germinal center is present. The intima and media of the blood vessels in the involved area may show thickening. The foreign body giant cell granuloma may persist from earlier states. Hashimoto's disease begins in a normal thyroid gland, whereas Riedel's struma may begin in an adenomatous goitre.

The third group, chronic nonspecific thyroiditis, encompasses those chronic inflammations of the thyroid without specific etiology and which are not associated with hyperplastic or adenomatous goitres. Grossly there is little change in the gland, although there is some fibrosis and usually

some increase in size. The affected areas are usually irregular in arrangement and involve only part of the thyroid. Adhesions may be present but are not dense.

Microscopically, there are localized areas of fibrosis with an infiltration of chronic inflammatory cells. The acini may show some degenerative changes in the involved areas, but adjacent acini are not affected. An occasional giant cell is present, but there are no granulomatous type lesions. An occasional focal accumulation of lymphocytes is present but rarely is there a true lymph follicle. The blood vessels are usually unaffected.

Thyroiditis as a disease associated with a hyperplastic or adenomatous goitre is fairly common and is often responsible for the symptoms that bring the patient to see a physician. There were 14 of these cases in our series of 172 thyroidectomies. Microscopically this type of chronic thyroiditis cannot be differentiated from a chronic nonspecific thyroiditis except for the presence of the associated goitre.

Ten of these secondary chronic thyroiditis cases occurred with nodular goitres and 4 occurred with hyperplastic goitres. Marshall *et al*[2] point out that in hyperplasia of the thyroid gland there is frequently an infiltration with lymphocytes and often the formation of secondary lymph follicles. A small amount of fibrosis often accompanies the lymphoid infiltration, particularly in the later stages of involution. In adenomatous goitre fibrosis of the gland owing to hemorrhage or colloid spillage is frequent.

SYMPTOMATOLOGY

The chief symptoms in Hashimoto's disease are diffuse enlargement of the thyroid gland, tightness in the neck, a sense of pressure in the neck, and hoarseness. Hoarseness may be marked at times. Occasionally these individuals will exhibit the symptoms of mild hyperthyroidism with nervousness and tremor. Again these patients may exhibit the signs and symptoms of myxedema with puffy facies, falling hair, dry coarse skin, fatigue, weight gain, and slowness of body movement.

In the authors' 4 cases of Hashimoto's disease 3 of the patients gave a history of hoarseness varying from several months to many years. All 4 had noticed enlargement of the thyroid gland. Two of the cases complained of a pressure sensation in the neck and 2 complained of nervousness. None of the 4 exhibited signs of myxedema when first seen, but 2 of the patients had a slight elevation of the basal metabolic rate above normal.

Riedel's struma causes more marked pressure symptoms in the late stages of the disease. Pain may be present at times. One of the authors' 2 cases of Riedel's struma had acute pain of several weeks duration prior to her first visit. Nervousness is a complaint and as suggested by Schilling[5] this is probably a secondary factor because of the anxiety state created by the dyspnea and choking sensations. The duration of symptoms in our 2 cases was twenty months in 1 and several years in the second case.

Chronic nonspecific thyroiditis may cause a sensation of tightness or pressure in the neck, depending on the stage of the disease. Hoarseness may be present as it was in the authors' 1 case. Mild hyperthyroidism may at times be manifested by nervousness and tremor of the fingers. The predominant symptoms in our single case were related to a mild hyperthyroidism. The initial basal metabolic rate was plus 37. This patient had been aware of enlargement of the thyroid gland for two years.

DIAGNOSIS

The preoperative diagnosis of chronic thyroiditis is not easy, as evidenced by the report of Marshall *et al*[2] of only 23.5 per cent correct preoperative diagnosis in these cases.

In our group of 7 cases of chronic thyroiditis the correct preoperative diagnosis was made in 3 cases, or 42.8 per cent. The greatest error was in the group of Hashimoto's disease, the correct preoperative diagnosis being made in only 1 of the 4 cases, or 25 per cent. The common error in struma lymphomatosa is confusion with an adenomatous goitre. This latter diagnosis is adhered to not only preoperatively,

but occasionally postoperatively by the operator.

If the examiner keeps chronic thyroiditis in mind while taking the history and during the examination the diagnosis of chronic thyroiditis should not be too difficult. A history of pressure in the neck and hoarseness should make one suspect the presence of a chronic thyroiditis.

Hashimoto's disease almost always occurs in women, usually of middle age. There is a bilateral symmetrical diffuse enlargement of the gland and the surface has a feel that has been adequately described as "pebbly-feeling." The gland is firm to the examining fingers and this should indicate to the examiner the possibility of thyroiditis. The so-called "pebbly-feeling" of the gland is often so marked that the diagnosis of adenomatous goitre is a frequent error.

The diagnosis in Riedel's struma is, as a rule, much easier to make than that of struma lymphomatosa. The marked pressure symptoms in the neck, plus the extreme hardness of the gland, should make the diagnosis obvious. This condition does not involve both lobes in a symmetrical enlargement, as does struma lymphomatosa. Riedel's struma is more common in women but does occur in males. Hoarseness may be a prominent symptom.

The differentiation of Riedel's struma from malignancy is difficult to make preoperatively and operation with biopsy should be recommended in these cases, if for no other reason than to rule out a malignancy, although operation for relief of pressure is usually necessary.

Hoarseness may be prominent in chronic nonspecific thyroiditis but is usually not as marked as in Hashimoto's disease or Riedel's struma. The gland in chronic nonspecific thyroiditis is as a rule slightly to moderately enlarged, with one lobe often being larger than the opposite. Pressure symptoms are often present, but not to the extent of Riedel's struma. The gland is firmer than that of a hyperplastic goitre and does not have the pebbly feel of Hashimoto's disease. Mild hyperthyroidism may be present.

TREATMENT

The treatment of Hashimoto's disease or struma lymphomatosa is bilateral partial thyroidectomy. This affords relief of the pressure symptoms which are a prominent feature of this disease. The hoarseness is not affected by the operation. These patients must be followed carefully postoperatively as the incidence of myxedema following surgery is high. All four of our cases of Hashimoto's disease developed myxedema postoperatively.

Other complications following operation are tetany and recurrent laryngeal nerve paralysis. None of these complications occurred in our 7 thyroiditis cases.

The treatment of Riedel's struma is operation. Operation is advised in order to relieve the pressure symptoms and also to rule out the possibility of malignancy of the thyroid gland. A conservative type of thyroidectomy is advisable in early Riedel's struma. This type of procedure was done in the authors' 2 cases. In far advanced Riedel's struma the isthmus only should be removed, as suggested by Lahey[6]. This latter procedure relieves the pressure and avoids the serious complications that might ensue if a bilateral thyroidectomy is attempted. These individuals must be examined frequently following operation, as myxedema is a common postoperative complication in all types of chronic thyroiditis. One of our 2 cases developed myxedema following a bilateral thyroidectomy.

The treatment of chronic nonspecific thyroiditis in most instances is surgical. Operation is advisable in most of these patients in order to relieve the pressure symptoms and to rule out malignancy. When the symptoms are minimal and the examiner is reasonably certain there is no malignancy present, conservative management may be justified. When there is doubt relative to the benign character of the lesion, operation should be done. A recent case seems to illustrate this point. A young woman, age 21, was seen because of a diffuse mass in the neck and a complaint of tightness in the neck. The thyroid gland was firm and both lobes were enlarged. A

diagnosis of thyroiditis was made, but because of the possibility of malignancy operation was advised. At surgery frozen sections were reported by the pathologist as malignant; therefore, a total thyroidectomy was performed. The final report was papillary cystadenocarcinoma.

Occasionally a patient with any one of these types of chronic thyroiditis will exhibit mild hyperthyroidism. In such instances propylthiouracil and Lugol's solution should be given preoperatively. Patients receiving propylthiouracil must be followed closely, as these individuals may become myxedematous after a relatively small dosage of the drug. The presence of myxedema is a contraindication to thyroidectomy and should be corrected before proceeding with surgery. Myxedema may be the cause of an obstructed airway if operation is attempted in the presence of the condition.

SUMMARY

A study of 172 consecutive thyroidectomies from the surgical services of the Mahorner Surgical Group at the Touro Infirmary and the Southern Baptist Hospital in New Orleans, Louisiana, reveals an incidence of 4.06 per cent of chronic thyroiditis. Out of the 7 cases of chronic thyroiditis, there were 4 cases of Hashimoto's disease, an incidence of 2.32 per cent; 2 cases of Riedel's struma, an incidence of 1.16 per cent, and 1 case of chronic nonspecific thyroiditis, an incidence of 1.74 per cent.

A study of all thyroid specimens removed surgically at the Southern Baptist Hospital during the period January 1, 1945, to April 1, 1949, revealed an incidence of chronic thyroiditis of 8.11 per cent. Out of the 18 cases there were 9 cases of Hashimoto's disease, an incidence of 4.06 per cent; 3 cases of Riedel's struma, and incidence of 1.35 per cent and 6 cases of chronic nonspecific thyroiditis, an incidence of 2.70 per cent.

The symptomatology, diagnosis, pathology, and treatment of acute and chronic thyroiditis are discussed.

CONCLUSIONS

After study of a series of 172 consecutive thyroidectomies, a study of the record room statistics at Touro Infirmary and the Southern Baptist Hospital, and a review of all thyroid specimens removed surgically at the Southern Baptist Hospital during the period January 1, 1945, to April 1, 1949, the authors conclude that chronic thyroiditis is far more common in the Gulf States than in other sections of the United States.

Although the authors have no figures for comparison, it is their impression that acute thyroiditis is also more common in the Gulf States than in other parts of the United States.

REFERENCES

1. McSwain and Moore: Struma lymphomatosa. Surg. Gynec. & Obst., 76:562, (May) 1943.

2. Marshall, S. F., Meissner, W. A., and Smith, D. C.: Chronic Thyroiditis. New England J. Med., 238:758, (May) 1948.

3. Boyden, Coller, and Bugher: Riedel's struma. West J. Surg., 43:547, (Oct.) 1935.

4. DeCourcy, J. L.: A new theory concerning the etiology of Riedel's struma. Surgery, 12:754, (Nov.) 1942.

5. Schilling, J. A.: Struma lymphomatosa, struma fibrosa and thryoiditis. Surg. Gynec. & Obst., 81:533, (Nov.) 1945.

6. Lahey, F. H.: Thyroiditis: Operative procedure for relief of tracheal constriction. Surg. Gynec. & Obst., 60:969, (May) 1935.

7. Parmley, C. C. and Hellwig, C. A.: Lymphadenoid goitre: Differentiation from chronic thyroiditis. Arch. Surg., 53:190, (Aug.) 1946.

8. Ficarra, B. J.: Struma lymphomatosa: Clinicopathologic study. Arch. Surg., 52:729, (June) 1946.

9. Hotz, R.: Struma fibrosa: Giant cell variant. Ohio State M. J., 43:1153, (Nov.) 1947.

10. Graham, A.: Riedel's struma in contrast to struma lymphomatosa. West J. Surg., 31:681, (Sept.) 1931.

11. McClintock and Wright: Riedel's struma and struma lymphomatosa. Ann. Surg., 106:11, (July) 1937.

CARCINOMA OF THE PROSTATE*

A REVIEW OF MODERN MEDICAL AND SURGICAL TREATMENT

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BALTIMORE

The treatment of carcinoma of the prostate can best be discussed from two aspects; first, curative methods, second, palliative methods. It is generally recognized that in spite of the often spectacular

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results which occur as a result of estrogen therapy in the treatment of this disease, there is as yet no cure reported in the literature, controlled by pretreatment biopsy and either operative or autopsy findings, in which a five-year cure of the disease has been accurately demonstrated. The same fact is true in regard to radium or deep x-ray therapy, which methods, of course, have been largely superseded by the far more effective hormone influences, the knowledge of which we owe largely to the fundamental work of Huggins.

It therefore seems safe to say that in the present state of our knowledge, complete surgical extirpation of the prostate in its surgical capsule is the only method which we at present have available for permanent cure of cancer of the prostate. It is evident that, as in cancer elsewhere in the body, to effect such a cure an early diagnosis must be made before the growth has extended too far for complete radical excision and before metastatic deposits have occurred.

Unfortunately, carcinoma of the prostate is insidious in its early stages. It has been emphasized that the neoplasm arises in the posterior lobe in over 80 per cent of cases, and slowly invades the lateral lobes, often extending into the fascia surrounding the seminal vesicles before symptoms of urinary obstruction appear. Although this fact will probably always lead to an inevitable diagnostic delay, in many cases until the growth has extended too far for complete radical excision; on the other hand, it remains as a challenge to our diagnostic acumen and to the thoroughness of our diagnostic measures.

DIAGNOSIS

The diagnosis or the suspicion of early malignant disease rests entirely on the palpation of a hard nodule in the prostate on careful rectal examination. It is not the purpose of this presentation to discuss the pitfalls of differential diagnosis, but it is, of course, self-evident that all measures should be undertaken to exclude prostatic calculi—which can be done by simple x-ray studies—tuberculosis of the prostate, and chronic prostatitis, the most common conditions with which cancer can be confused. In spite of the most careful studies there will always remain a certain number of cases in which the diagnosis can only be absolutely confirmed by perineal exposure of the gland and a frozen section biopsy. In the Brady Urological Clinic we have not practiced needle biopsy, possibly because we have never had sufficient confidence in our cytological experience, and furthermore, in this procedure there will always remain the doubt that the needle has obtained a specimen from the particular area under suspicion. We have in the past few months made some cytological examinations of expressed prostatic secretion with a modification of the Papanicolaou method, but the results have given us little help, possibly on account of the small series studied and our own inexperience. However, with a knowledge of the anatomy of the prostate in mind, it would appear to be extremely optimistic to expect to extrude by rectal pressure through normal lateral lobes a sufficient number of malignant cells to make a positive diagnosis from a small neoplasm limited to the posterior capsule.

The possibility of dissemination of tumor cells by manipulation of a neoplasm also arises. This question will always remain difficult to determine, if not insoluble; but if tumor cells can be dislodged in sufficient numbers to be recognizable in the expressed secretion, it is certainly possible, if not probable, that these free neoplastic cells might also invade the perineural lymphatics. Certainly the general surgeons would hesitate to massage a suspicious lesion of the breast to obtain material for microscopic study, and urologists, I am sure, would not consider it justifiable to exert pressure on a kidney tumor for the purpose of expressing cells which might be recognized in the urine; and certainly any undue manipulation of a testicular tumor has always been severely condemned.

It has therefore been our custom, when a tentative diagnosis of early carcinoma has been made, to inform the patient that

malignant disease is suspected and that if the diagnosis is corroborated by frozen section biopsy a radical operation will be done. With the patient prepared for the operation, the suspicious area in the posterior lobe is exposed in the usual manner, a generous biopsy obtained, frozen sections studied and if positive evidence of malignant disease is evident, the radical operation is forthwith carried out. If no evidence of cancer can be demonstrated in several satisfactory sections, the site of the biopsy is thoroughly coagulated, a retention catheter introduced and the wound closed.

The time consumed in making biopsy studies may vary considerably, but is approximately twenty minutes. We have found that this time can be very profitably used in thorough electrocoagulation of the site from which the biopsy was obtained and in complete mobilization of the prostate laterally until the blades of the tractor in the bladder can be easily felt, and posteriorly until the seminal vesicles can be palpated as far up as their upper extremities. Time thus spent in thorough mobilization will greatly expedite the subsequent steps of the operative procedure.

SELECTION OF PATIENTS

Careful selection of cases suitable for the radical operation is essential if morbidity and mortality are to be avoided and satisfactory postoperative results obtained. Certain criteria must be emphasized although they may be somewhat elastic. First, the obvious malignant induration must not extend beyond the capsule of the gland, into the membranous urethra, or extensively involve the fascia around the seminal vesicles, and the whole gland must be freely movable. Second, there must be no demonstrable metastases, either on physical examination or more particularly by x-ray studies, and the acid phosphatase determination should be within normal limits. Third, the patient should be a good surgical risk and, most important, his life expectancy must be good.

This latter point, we feel, is of paramount importance. Prostatic cancer is a slowly progressing disease in most cases and we cannot condemn too strongly the performance of the procedure in an elderly person whose life expectancy is obviously limited. In older individuals, generally speaking, muscle tone is to some degree at least impaired and the functional results following the operation will never be as satisfactory as when the procedure is carried out on younger men, more robust, and with good muscle tone. The criticisms which have been directed at the radical operation emphasizing unsatisfactory postoperative results, we feel are largely due to results in cases which should never have been subjected to the procedure. From the foregoing it has therefore been our custom to reserve the radical operation for those patients under seventy years, but the rule is by no means didactic as exceptions will always be found on either side of this limit.

The original concept of Hugh H. Young, who first devised and carried out the operation, was the complete removal of the prostate with the seminal vesicles and the fascia surrounding these structures up to their extremities. It is in this fascia and not in the vesicles themselves that extension of the neoplasm will be found if careful microscopic studies are carried out. Some writers have advocated various modifications of Young's original operation, including splitting of this essential fascia to isolate each vesicle separately, and even to divide the anterior commissure of the prostate itself to facilitate the operative procedure. By this technic the integrity of the fascia is violated and a true radical operation is not carried out.

RADICAL OPERATION

We feel very strongly that if the surgeon decides that an early carcinoma of the prostate is present and that the criteria, without which the radical operation should not be undertaken, are satisfied in the particular case under consideration, the most complete operative removal which is technically possible should be carried out. As an analogy, when general surgeons make a diagnosis of carcinoma of the breast, certainly no compromise is made and the most complete excision which is technically possible is performed.

No discussion of the technic of the operation will be presented except to emphasize the fact that after the excision has been completed great care must be taken in placing the sutures which unite the stump of the membranous urethra to the neck of the bladder. We have found that by employing Vest's principle in bringing these sutures out through the soft tissues of the perineum, an accurate approximation can be accomplished. In addition, this technic prevents the possibility of tying the knots directly over the external sphincter muscles, thus endangering their integrity with the possibility of resultant incontinence.

The radical operation entails no greater dangers than perineal prostatectomy for benign hypertrophy; in fact, the mortality is less. This can be explained by the fact that the radical operation is only undertaken in good risk patients in the younger age brackets, whereas perineal prostatectomy for benign hypertrophy is often undertaken in older, poor risk patients in whom the operator's purpose has been to perform a quick, nonshocking operation in the shortest time possible. During the past ten years 186 radical operations have been performed by various operators in the Brady Clinic with 6 deaths. In the last 100 cases there were 3 deaths, in the last 50 none. In a personal series of 70 cases there has been no mortality.

Spinal anesthesia is the method of choice. The position of the patient on the table is sometimes an ordeal and pentothal sodium is often administered when there is too much discomfort. Hemorrhage is never a serious problem. The key to this lies in the careful exposure and ligature of the lateral ligaments, and ligation of the tissues above the tips of the seminal vesicles. When the bladder wall in close proximity to the prostate is divided, bleeding vessels are often visualized and can easily be controlled, and following the anastomosis of the stump of the urethra to the anterior aspect of the bladder defect careful closure of this defect will control all intravesical bleeding. If oozing occurs from the depths of the operative site it can easily be controlled by oxycel gauze, but we have found this rarely necessary.

In the older days of the development of the operation in several cases the ureters were obstructed, probably being included in the sutures used to close the bladder defect. With the excellent relaxation afforded by spinal anesthesia when the prostate in its capsule is excised from the neck of the bladder, a good visualization of the trigone and ureteral orifices can be obtained with adequate retraction and the integrity of these structures easily preserved. The intravenous injection of indigocarmine is of great help in recognizing the ureteral orifices and insuring their integrity.

Injury to the rectum has not occurred in the last ten years. Such an unfortunate complication would most probably occur during the exposure of the prostate before the urinary tract has been opened and should be easily recognized. Although I have never been faced with this complication, if it should occur the injury should be carefully repaired and the operation completed in the usual way, relying on the urethral catheter and the integrity of the rectal closure to prevent the development of a recto-urethral fistula.

The indwelling urethral catheter is allowed to remain about ten days, during which time the perineal wound will completely close in most cases, but occasionally persistent perineal leakage will necessitate the reintroduction of the catheter. Following removal of the catheter incontinence will persist for a variable interval of time, but complete control is usually reestablished within a month or less after the patient's discharge from the hospital. If there are any evidences of obstruction, manifested by difficulty, or a stream of small calibre and poor force, a careful dilatation of the urethra should be carried out, and in cases where incontinence is present this procedure will often result in great and immediate improvement in the functional result. In properly selected cases and with proper operative technic complete and permanent urinary incontinence does not occur.

The fact that, as a result of the radical

operation, sexual function is seriously impaired, if not totally abolished, must be faced frankly and patients should always be informed of this possibility. In our experience with most intelligent patients the possibility of complete excision of a malignant disease outweighs interference with sexual function. Following the operation it is now our custom to continue indefinitely the administration of estrogen in small doses in the hope that, if malignant cells have been left behind, they may undergo necrosis under this treatment.

SURVIVAL RATE

Several studies have been carried out (Young, Lewis, Colston and others) on the five year survival rate of patients subjected to the radical operation. These studies are in agreement that this rate is approximately 50 per cent.

Jewett has recently completed the first comprehensive study of the ten year survival rate, and in doing so has examined the clinical records and the specimens of 222 cases operated on in the Brady Clinic. It has been the custom in our clinic to record the impression of the examining surgeon's findings on rectal examination by means of a simple chart. Jewett's studies of these records and rectal charts convinced him that in many cases subjected to the radical operation there had been extracapsular extension of the growth before operation. He, therefore, in his study divided the cases into (a) those which showed no evidence of extracapsular extension on rectal examination and (b) those cases in which such extension was evident clinically. In Group (a), those with no demonstrable extracapsular extension, there was a ten year survival of 28 per cent, and "no patient who lived ten years without demonstrable recurrence of metastases subsequently showed evidence of cancer." This is, of course, a most significant finding. In Group (b) in which extracapsular extension was demonstrable, 7 patients lived six to nine years without evidence of cancer.

The question of local recurrence after the radical operation brings up some interesting points. Such recurrences become evident first between the bladder and the rectum so that they do not interfere with urinary fuction. They must arise from neoplastic cells which had invaded the fascia around the vesicles and before operation had extended into the fascia above the upper limits of these structures so that necessarily, because of technical limitations, they were left behind at the time of operation. As a rule, these cellular elements grow very slowly and have a tendency to extend upward, so that the site of the anastomosis between the stump of the urethra and the neck of the bladder is rarely, if ever, involved. We have no record of any case subjected to the radical operation in which subsequent urinary obstruction has developed due entirely to malignant recurrence. When recurrence after this operation does occur, death is due to the gradual extension of metastasis rather than to the result of urinary obstruction.

George G. Smith has been one of the most emphatic supporters of the radical operation, believing that it should be carried out in cases in which, on account of the extracapsular extension of the disease, there would seem to be no hope of a permanent cure. By this procedure the main mass of the disease is removed and there is no probability of subsequent operations necessary for the relief of recurring obstruction. It must be understood that the operation should not be undertaken when the gland is fixed or when extension too far into the fascia around the vesicles has occurred on account of the fact that under these circumstances the technical difficulties will be great and the subsequent functional result doubtful.

PALLIATIVE TRANSURETHRAL RESECTION

The introduction of transurethral resection for the relief of obstruction in inoperable prostatic cancer has been one of the greatest advances in the care of sufferers from this disease. Before this operation was perfected many of these patients were doomed to a permanent suprapubic cystostomy for the remainder of their lives. The only alternative to this palliative procedure was conservative perineal prostatectomy in

which the objective was to expose the gland by the perineal route and to remove by enucleation as much as possible of the obstructing malignant tissue. Often on account of the absence of line of cleavage between the posterior capsule and the obstructing tissue, sharp dissection and the use of a curette were found necessary. Inevitably, functional results were in some cases unsatisfactory with an occasional persistent perineal fistula, and varying degrees of incontinence persisted in some cases. Also, fixation of the gland to the rectum often made exposure difficult and injury to this structure was often a hazard. However, we considered then that conservative perineal prostatectomy gave far better results for the comfort and well being of the patient than its only alternative, a permanent suprapubic tube.

Transurethral resection has now almost completely superseded both conservative perineal prostatectomy and suprapubic cystostomy. The immediate results are usually most satisfactory and often brilliant, but in many cases, due to the fact that the neoplastic tissue around the neck of the bladder can never be completely removed by this procedure, recurrence of the obstruction, with the necessity of another resection, frequently occurs. In some cases satisfactory healing of the resected area never takes place, so that open ulcerations persist, followed by phosphatic incrustations with the resulting distressing symptom-complex of frequency, strangury and terminal hematuria which may make life a burden to the unfortunate sufferer. These sequelae can be avoided for the duration of the patient's life if surgical removal of all neoplastic tissue around the neck of the bladder is technically possible, even though a permanent cure cannot be expected.

REPORT OF CASE

It is always dangerous to quote an individual case, but in this connection the case of D. McM. comes to mind. This 60 year old man was seen in 1926 with increasing urinary symptoms. On examination a moderate grade of prostatic hypertrophy was found, but in the posterior capsule there was an elevated nodule, stony hard in consistency. A diagnosis of benign prostatic hypertrophy with a circumscribed area of malignant disease in the posterior capsule was made and a radical perineal prostatectomy was done. Convalescence was uneventful except for temporary incontinence, and a few weeks after leaving the hospital he had regained complete control, which continued so throughout the remainder of his life. Study of the operative specimen showed benign prostatic hypertrophy with an area of well differentiated adenocarcinoma, which on microscopic study had invaded both lateral lobes and had extended into the fascia around the base of each seminal vesicle, the involvement being more pronounced on the left side.

He was seen at yearly intervals and no evidence of recurrence was detected until four years after operation when an indurated nodule, which was considered a definite recurrence, was palpated high up on the left side between the posterior bladder wall and the rectum. This nodule increased in size slowly through the years; there was never any interference with bowel function and he always had excellent urinary function with a normal bladder capacity, no residual, and perfect control both day and night. He died of coronary occlusion in 1940, fourteen years after operation, always retaining perfect urinary control and with no disturbance of bowel function.

Through the courtesy of the physician who performed the autopsy the genito-urinary tract and the rectum were made available to us for study. Between the left posterior bladder wall and the rectum there was an irregular mass measuring about 6 by 4 cm. This mass was adherent to both bladder wall and rectum, but had not invaded either of these structures, which explained the fact that there had been no interference with either urinary or rectal function. Microscopic studies showed the mass to consist of well differentiated adenocarcinoma similar in structure to the original tumor in the prostate excised fourteen years previously. In all probability this mass had grown slowly from cells which had been present in the fascia around the vesicles at the time of the original operation lying above the upper limits of the original procedure.

Had this patient been subjected to transurethral resection when he was first seen an appreciable amount of malignant tissue must inevitably have been left around the prostatic orifice, with recurrence of obstructive difficulties and further resection for relief inevitable. It is inconceivable that, had transurethral resection been chosen as the procedure of choice when the patient was first examined, he would have had a life span of fourteen years with normal urinary function and control, and no evidence of infection.

HORMONE THERAPY

Cancer of the prostate and metastatic growths from the original tumor have been proved to be dependent for their growth on androgen stimulation, and the fundamental

research of Huggins has demonstrated that when the effect of this hormone has been repressed, either by orchiectomy or the administration of estrogen, in approximately 80 per cent of cases there will be a regression of both the original neoplasm as well as the metastases. Kahle and others have described the microscopic changes which occur in the neoplastic cells thus deprived of androgen stimulation, the chief manifestations of which are pyknosis of the nuclei, rupture of cell membranes, disintegration of malignant cells and necrosis.

Clinically, the results of elimination of androgen stimulation are manifested by marked regression of the neoplasm on rectal examination, often with complete disappearance of the induration in the fascia surrounding the seminal vesicles, so that in some cases all evidence of third degree induration will disappear and a diagnosis of malignant disease could no longer be made by the findings on rectal examination. Metastatic lesions will often show remarkable changes by x-ray examination and in some cases extensive lesions in bone will often completely disappear, apparently to be replaced by regeneration of normal bone structure.

The necrosis of malignant cells due to deprivation of androgen has been postulated by Huggins as due to interference with the enzyme mosaic of the individual cells. This cellular necrosis is most pronounced in the peripheral areas of the tumor where the malignant cells are younger and grow more actively and are therefore more dependent on androgen stimulation.

From these considerations it seemed possible that neoplasms which on first examination had progressed too far for complete extirpation by radical operation might, on estrogen therapy, regress to such an extent that the radical operation could be successfully carried out. On this account it has been our custom when a diagnosis of cancer of the prostate has been made to commence immediately the administration of estrogen therapy by means of diethylstilbestrol. In cases which on examination seem suitable for radical operation, it has seemed to us that this therapy will cause death of some of the malignant cells at the periphery of the tumor so that the chance of a permanent cure will be definitely enhanced. In cases which seem on first examination to be too advanced for radical operation, regression of the neoplasm might occur to such an extent that the operation could be rendered technically possible in a case, which without this therapy would be considered inoperable and therefore hopeless.

A small series of 7 cases has been reported which on first examination showed that the neoplastic growth had progressed outside the capsule or far up in the fascia around the vesicles, so that the possibility of a cure by radical operation seemed obviously impossible. Under stilbestrol therapy the findings on rectal examination changed to such an appreciable degree that the operation was subsequently carried out in all these cases without technical difficulties. Microscopic studies of these operative specimens showed that while degenerative changes and necrosis of many malignant cells were evident, viable cells could usually be found in the fascia around the vesicles. All these cases were operated on four years ago or longer, and a follow-up report on them will be made when the five-year interval has elapsed for them all. At present writing one patient died within a year from operation of recurrence and metastases. One died three years after operation of cancer of the sigmoid which bore no relation to the original tumor, no evidence of which was clinically evident, but no autopsy was obtained. Four patients are alive, but have definite clinical evidence of recurrence, but have normal urinary function. One patient is alive and well three years and six months after operation with no clinical evidence of recurrence or metastasis.

This short series of cases which would have been considered inoperable before the advent of estrogen therapy on account of the local extent of their disease, were operated upon in the hope that a cure might be obtained in some of them, and also in the hope that their life expectancy might be

increased by the removal of the main mass of their malignant disease.

In those cases of extensive carcinoma of the prostate which do not regress sufficiently to justify performance of the radical operation, it has been our custom to continue estrogen therapy either with diethylstilbestrol, 1-2 mgm., daily, or ethinyl estradiol, 0.15 mgm., daily. These dosages are almost invariably well tolerated although, of course, some hypertrophy of the breasts with initial hypersensitivity may be expected. Larger doses of estrogens have been advised by some writers, but in our own experience we have not been able to observe any more favorable response with higher doses than with the smaller ones which we customarily employ. The only way to settle this question would be to have under careful observation a series of cases under small estrogen dosage. Inevitably, at varying intervals of time the estrogen treatment will begin to lose its inhibiting effect as evidenced by progression of the prostatic neoplasm and growth of metastases with pain. At such a time, if higher estrogen dosage could be shown to result in another regression of the local lesion and regression of the growth of the metastases with relief of pain, the case for higher dosages would be sustained. However, we know of no sufficiently well controlled series of cases in which this sequence of events has been clearly demonstrated.

It is not uncommon, of course, to see a case of advanced carcinoma of the prostate, with or without demonstrable metastases, with the presenting symptoms of acute retention. Estrogen therapy is immediately instituted and a retention catheter introduced. After a week or ten days the catheter is removed, and in some cases it will be found that sufficient regression of the neoplasm has occurred so that the obstruction is relieved and relatively normal urinary function may be regained and retained over an appreciable interval of time. This method is preferable to immediate transurethral resection on account of the fact that, with the growth of the neoplasm, obstruction will recur and multiple resections be necessary, followed by inevitable infection.

ORCHIECTOMY

It has been our custom to reserve orchiectomy for these cases in which it has become evident that estrogen control of the disease has become ineffective. It is true that there is definite evidence that orchidectomy performed after a patient has been on stilbestrol therapy is often ineffectual and rarely gives the spectacular results which are so often observed when the operation is carried out without previous estrogen therapy. Nevertheless, in many cases treated by estrogens when it becomes evident that their effect in controlling the disease has begun to fail, orchiectomy in some cases at least will lead to definite alleviation of symptoms, although this is only temporary.

Conversely, when clinical evidence of progression of cancer of the prostate and its metastases become evident at varying intervals of time after orchiectomy has been previously performed, it is rare that the progress of the disease can then be favorably influenced by the institution of estrogen therapy.

Careful analyses of large series of patients with inoperable cancer of the prostate must be studied before definite conclusions can be reached as to the relative value or orchiectomy and eserogen therapy and the time that either one or both of these measures should be instituted. Creevy has advised that endocrine therapy should not be started until symptoms appear. He then gives estrogen therapy and when it is evident that this therapy has become ineffectual, an orchiectomy is performed. When an agent which is effective against cancer is known and its success has been so definitely proved, as is the case with endocrine therapy in carcinoma of the prostate, it would seem to be a mistake, at least on theoretical grounds, to withhold its use until the neoplasm had progressed to a sufficient degree to produce symptoms, even though sufficient statistical studies are as yet unavailable to support this point of view.

In the care of patients with far advanced

cancer of the prostate in whom estrogen therapy and castration have been proved ineffectual in the control of the disease, deep x-ray therapy will in some cases result in temporary palliation; and in rare cases where suffering from bone metastases has become unbearable, subarachnoid alcohol injections, or the administration of novocaine subdurally according to the method of Hingson, or finally cordotomy may give the patient sufficient relief to make his last days tolerable.

SUMMARY

A review of our present knowledge of carcinoma of the prostate has been presented with especial emphasis on the treatment of this disease. The great progress in the palliative treatment of this condition as a result of the institution of endocrine treatment, either by orchiectomy or administration of estrogens, has been discussed. In spite of the great alleviation of human suffering which has been accomplished by these very great advances in therapy, there is at present no case of permanent cure of cancer of the prostate by endocrine therapy recorded in the literature.

The radical operation offers us at present our only hope of complete eradication of this disease, and follow-up studies have shown that in properly selected cases subjected to this procedure there will be a five-year survival rate of approximately 56 per cent, and more significantly a ten year survival rate of 28 per cent.

The difficulties in early diagnosis have been discussed and the challenge that this very common disease, curable in its early stages, presents, has been emphasized.

No bibliography will be appended because the foregoing presents the personal views of the writer. The writings of Young, Geraghty, Bumpus, Hinman, and more recently Huggins, Nesbit, Vest, Creevy and Jewett, to mention only a few of those authors who have contributed to the literature on cancer of the prostate, are readily available.

THE RETROPUBIC APPROACH TO VESICAL OBSTRUCTIONS

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Another era of prostatic surgery was established when Millin demonstrated his method of retropubic prostatectomy in 1945. One's first reaction agrees with W. K. Irwin's who stated that fashions change in the field of prostatic surgery with almost the same bewildering frequency as in that of dress. In actuality of course this is not true for prostatic surgery has progressed in an orderly fashion always being limited by the currently available means of controlling postoperative infection and bleeding.

The retropubic approach or anterior extravesical approach to the prostate through the space of Retzius is not a new concept. Basically this operation was suggested by Zucherkandl in 1906, by Van Stockum in 1909 and in 1922 by Lidski. Jacobs and Casper stirred the Western Section of the American Urological Association with their "prevesical prostatectomy" in 1933. For more than forty-three years this operation had been attempted and received intermittent attention but without a sufficient following or success to warrant adoption. Millin introduced a grossly simplified procedure which literally defied the accepted surgical dictum that the space of Retzius would not tolerate ingression. The sulfonamides and the antibiotics have bolstered the less adventurous surgeons.

We are not of the opinion that the retropubic operation is the universal answer to prostatectomy, but we feel that it has a place in the relief of certain instances of prostatic obstruction. This dissertation will present our experiences with retropubic surgery which include the conservative and radical methods of retropubic prostatectomy, and in addition, the relief of poster-

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ior urethral and vesical neck obstructions in children.

The retropubic prostatectomy employs an extravesical approach to the prostate which, though seemingly infrequently appreciated, is an extravesical structure. There is an additional advantage to the capsular incision which has not been given adequate consideration in the literature. This factor is the relatively greater blood supply of the prostatic capsule as compared to the bladder wall. To appreciate this point one needs only to glance at the illustrations of Farabeuf and Tsaknis who studied the blood supply of the bladder and rendered a most detailed anatomical description in 1905. The rapidity of capsular healing has been spectacular on the two occasions which we have studied. An aged individual developed a large strangulated vesical diverticulum following retropubic prostatectomy which defied catheter drainage. On the fourth day after prostatectomy it was felt that surgical interference was imperative to establish drainage but it was impossible to find the capsular wound. Hence, instead of draining the diverticulum through the capsular wound it was necessary to undertake a cystotomy. The second patient, who had a history of severe bleeding following other operations, developed a massive postoperative hemorrhage into the bladder. Although this occurred on the third morning following surgery, the capsular wound could not be defined and a cystotomy was used to effect vesical drainage. It was readily seen in these two instances that the capsular wound was firmly healed within seventy-two hours after surgery; and in the second patient not only was the capsular wound not readily visible or palpable but it withstood a very firm gauze pack which was used to control an extremely active hemorrhage from the vesical neck. This rapidity of capsular healing is a factor of great merit.

RETROPUBIC PROSTATECTOMY

In our opinion this operation is indicated in all instances of benign prostatic hyperplasia of medium or large size. We reserve the transurethral resection in nonmalignant lesions for the small glands, fibrous vesical neck contractures, and fibrous prostates with obstruction. The transvesical or suprapubic prostatectomy is employed only in giant vesical calculi associated with prostatism and here only if the calculus measures by x-ray more than 4 cm. in diameter. In instances where a cystotomy has been necessary we still execute a retropubic prostatectomy and have been justified in every patient by a most benign operative and postoperative course. Although the perineal prostatectomy was our first love, we have abandoned this route except for an occasional demonstration or for old time's sake.

In the retropubic prostatectomy the prostate is exposed through either a longitudinal or transverse suprapubic incision which includes both the skin and rectus fascia. The rectus muscles are separated, the hypogastric fascia opened and the empty bladder displaced cephalad to expose the variable vascular endopelvic fascia overlying the prostate. Small gauze packs are inserted on either side of the prostate to facilitate prostatic delineation. If the veins are prominent they are individually doubly ligated and severed. If the endopelvic veins are not large they are disregarded and dealt with individually when bleeding ensues following incision of the prostatic capsule. It has been our practice to introduce a subcapsular suture at the apex of the bladder coursing across the anterior surface of the capsule to diminish bleeding from the capsular veins. We believe this step is an adjunct and were interested to note that Millin employs a similar technic which was reported in a recent account of one of his operative clinics.

After the prostatic capsule has been gently freed of its attached tissue the shining capsule is visualized. We then inject the adenoma, as Millin has advocated with a solution prepared by mixing 15 cc. of 1 per cent procaine with an ampoule of pitressin. Into each lateral lobe, with a needle point just below the capsule, an 8 cc. increment of this solution is injected. This definitely reduces bleeding during the period of enucleation and further it acts as

a means of hydraulic dissection of the adenoma from the false capsule. We have employed a variety of mixtures for this injection and are at a loss to explain why the procaine-pitressin solution is more effective than a saline-pitressin mixture.

The prostatic capsule is incised transversely at approximately 1.5 cm. distal to the vesicoprostatic junction. The length of the incision is dependent upon the size of the prostatic adenoma and is long enough to facilitate an atraumatic enucleation. All layers of the capsule are divided with a single stroke of the knife and the bulging adenoma can be seen in the opening of the capsular wound. Bleeding from capsular veins at this point may be brisk and adequate visualization is easily afforded by the use of suction. The individual veins are clamped and sutured for positive hemostasis. The operation as practiced by Millin is conducted entirely by electrocoagulation; however, we have not experienced similarly satisfactory hemostasis. It is to be recalled that these prostatic veins are actually venous channels which have a sparsity of muscle fiber and their attachment to the surrounding capsular tissue may defeat the usual effectiveness of electrocoagulation.

Following capsular hemostasis the prostatic adenoma is separated from the false prostatic capsule with scissors point dissection throughout its entire anterolateral circumference, and before delivering the gland into the retropubic space the posterior urethra is severed with scissors at the apex of the adenoma. When the gland is delivered from the capsule it is elevated as much as is possible in order to visualize its attachment to the vesical neck. The prostate is now attached to the bladder only by a mucosal cuff and this is cut across. The adenoma is removed and one can, for the first time, get a glimpse of the bladder cavity through the vesical neck. This entire procedure has been extravesical in both the approach and enucleation of the adenoma.

The prostatic arteries at the prostatovesical junction are sought and ligated with a figure of 8 suture. These arteries normally enter the prostatic capsule at its junction with the bladder at the levels of 5 and 7 o'clock. If the arteries cannot be visualized, we usually place sutures at these levels. The vesical neck is then inspected for small bleeders which are lightly fulgurated. After the vesical neck is free of bleeding points a catheter is introduced through the urethra which transverses the prostatic fossa and enters the bladder through the vesical neck.

The prostatic capsule is closed using a continuous suture of No. 1 chromic catgut encompassing all layers of the capsule. The suture to control any postoperative capsular ooze must be started and terminated well beyond the angles of the capsular wound. All layers of the capsule must be closed since any redundant capsular edge is a potential source of bleeding postoperatively.

After a small drain is left in the space of Retzius the abdominal wound is closed in layers and the skin is closed. Vesical drainage is accomplished by the urethral catheter alone. A bilateral vasectomy is done to prevent postoperative epididymitis.

The wound drain is removed on the second postoperative day and the urethral catheter is removed on the fourth day following surgery. The urine will most often be of burgundy color for the first twelve to eighteen hours, the color of strong tea the second day, and clear on the third morning.

We have tabulated below a resume of our first 234 retropubic prostatectomies which are compared to 500 other methods of prostatectomy, the majority of which were done by the perineal route by the late Owsley Grant. As can be seen at a glance the results are comparable, as might well be ex-

TABLE I

	234 Retropubic Prostatectomies	500 Open Surgical Prostatectomies	
Average Age	68.3	67.1	years
Total Period of Hospitalization	17.1	31.0	days
Postoperative Hospitalization	12.4	21.8	days
Catheter Period Postoperatively	6.2	11.6	
Mortality	3.1	3.9	%

pected, since both procedures are extravesical methods of prostatectomy. It is to be noted further that some of the apparent improved figures in the retropubic group could well be occasioned by the fact that antibiotics and sulfonamides were available.

The mortality of 3.1 per cent in the retropubic group constituted 7 patients all of whom died within fourteen days postoperatively. One died on the fifth day of an acute coronary thrombosis; another, 78 years of age, had an uncontrollable cardiac decompensation and bronchopneumonia and died a cardiac death on the thirteenth day following surgery. A man of 66 years died of an overwhelming perivesical infection which had presumably been present, though undiagnosed preoperatively. He is considered as an ill chosen subject for prostatectomy. The patient would not tolerate either urethral or cystotomy drainage since both had been used in an attempt to avoid operation. The remaining four patients of 71, 69, 59 and 77 years died on the seventh, eleventh, thirteenth and sixth days postoperatively from bronchopneumonia, cardiac failure, uremia, and an infarct, respectively.

The surgical complications which prolonged hospitalization in the series of retropubic prostatectomies are noted below.

TABLE II

Complication	Percentage Incidence	Number of Fistula Patients
Suprapubic fistula temporary	0.8 %	2
Wound infection	4.7 %	11
Secondary hemorrhage from the prostatic bed	2.05 %	6
Osteitis pubis	0.8 %	2

Suprapubic fistula occurred in 2 patients. The first was a severe diabetic who presented a wound infection and a most difficult wound healing problem; the duration of the fistula was twenty-one days. The subsequent course of the patient has been excellent. The other patient was suffering with a huge, narrow mouthed diverticulum which could not be resected because of the patient's generally poor state. The fistula healed on the twenty-seventh postoperative day.

Wound infection was recorded in any patient where there was induration of the wound and possibly some drainage thus causing the patient to be maintained in the hospital beyond the usually anticipated day of discharge. The longest period of hospitalization in this group was eighteen days.

Secondary hemorrhage from the prostatic bed occurred in 6 patients. Four of the group were so stricken during the period of hospitalization, and it was thought that the hemorrhage was due to bleeding from the prostatic arteries which had not been ligated. In two instances the bleeding was controlled by endoscopic methods employing electrocoagulation. A third patient suddenly developed moderate though persistent hemorrhages the day following removal of the catheter. The bleeding was finally controlled by traction alone. The fourth patient had a vesical hemorrhage on the third day after surgery while the urethral catheter was still in position. The bladder filled with clots and an immediate cystotomy became a life-saving measure. The source of the bleeding seemed generalized from the vesical neck and it was instantly controlled with gauze packs. In each of the above patients the remainder of the hospital course was uneventful. The remaining 2 patients suffered secondary hemorrhage following the seventh postoperative day; 1, on the day of discharge; this was controlled by a Foley bag. The other hemorrhaged on the eighteenth postoperative day, three days following discharge from the hospital, and this was restrained by electrofulguration cystoscopically. Secondary hemorrhage from the prostatic bed is a hazard of prostatectomy from which no method thus far has been immune.

There were two instances of osteitis pubis, both of which continued for approximately six months. One patient was a hugely obese, diabetic whose convalescense was delayed by poor wound healing. The other patient's convalescence was uneventful until the third week postoperatively

The appearance of osteitis pubis in the literature in connection with this operation seems to have been all too frequent and this constitutes certainly a most valid criticism. It is to be remembered, however, that the syndrome may well have been not so clearly recognized in the past and many instances may have gone undiagnosed and thus unreported following other methods of prostatectomy. We have seen one instance following perineal prostatectomy and recently Rosenberg and Vest reported two instances following transurethral resection.

RADICAL RETROPUBIC PROSTATECTOMY

The approach to the prostate in radical retropubic prostatectomy is identical with that used in the conservative operation. After the gland has been exposed and the paraprostatic fossa on either side delineated, the finger is inserted laterally at the apex of the gland and with gentle dissection the finger is passed as far as possible around the apex of the prostate. After the prostatourethral junction has been encircled a ligature is passed around the apex of the gland. The prostatourethral junction is distinctly palpable due to the firmness of the previously introduced No. 16 F. Nelaton catheter. The ligature is now tied tightly distal to the prostatic apex. This ligature compresses the dorsal veins entering the plexus of Santorini and materially reduces the bleeding upon division of the urethroprostatic junction. This step is best accomplished with a long scissors rather than a knife dissection.

After the prostate has been severed from the urethra, the urethral opening in the prostate is obliterated by applying a Lahey forceps to the apex of the gland to close the urethra and assist in elevating the prostatic apex in the dissection of the gland from the posterior structures. The prostate is elevated and freed from the rectum by blunt dissection. It is to be noted at this time that unless particular layer dissection is practiced it will be found that Denonvillier's fascia will adhere to the prostate and the rectum alone is displaced. We have not made any attempt to preserve a layer of Denonvillier's fascia over the rectum.

As the prostatic apex is elevated further, prostatic pedicles containing arteries to the gland are demonstrated. The pedicles are doubly clamped, tied, and severed. Following this the seminal vesicles covered by Denonvillier's fascia come into view. Denonvillier's fascia is cut across at the base of the prostate. The vasa deferentia are visible, and lateral to these structures lies the proximal portion of the vesicles. The vasa are dissected free and after a 5 cm. length has been obtained they are severed between ties.

The seminal vesicles are now dissected free to their distal portion and in this adherent tissue are small vessels to the vesicles. This tissue is doubly clamped, ligated, and severed.

The prostate is cut free of the bladder by opening the bladder at the anterior margin of the prostatovesical junction. The incision is continued around the entire base of the prostate, and the gland, with the attached vesicles and vasa deferentia, is removed.

Bleeding points on the vesical neck, which are usually few, are fulgurated and the bladder neck is attached to the urethral stump with sutures. Soon after the prostate is cut free of the urethra there are discovered most often bleeding points on the posterior urethral cuff. These bleeding points occur at the positions of 5, 6, and 7 o'clock. The bleeding vessels are controlled with transfixion sutures which are left long. These sutures are now used as the inferior sutures in attaching the bladder to the urethra. Before the sutures are tied at least two additional sutures are placed at the levels of 10 and 2 o'clock. A No. 22 Foley is passed through the urethra into the bladder.

After the sutures have been tied and the bladder neck has been approximated to the urethral stump, the residual opening in the bladder is closed with interrupted sutures so that the vesical outlet approaches the caliber of the urethra. A final mattress suture is now taken which approximates the residual edges of the bladder and then penetrates the urethral stump at 12 o'clock

position.

The abdominal wound is now closed in layers and a small rubber drain is left in place for two days. The urethral catheter is removed on the seventh postoperative day. In some of our earlier patients we were troubled with prolonged stress incontinence, but this has not occurred in later patients since greater care has been used in placing the urethral sutures and in utilizing the ligature sutures to minimize the number of sutures in the urethral stump.

We have executed a total of 13 radical retropubic prostatectomies, all but one being for carcinoma. Our criterion for the radical procedure is a clinical diagnosis of early carcinoma of the prostate without glandular fixation particularly at the apex. We have also employed the radical operation with excellent results in a firm fibrous prostate associated with severe subjective irritation. The results with this operation have been gratifying and we feel that its merit lies in the easily visualized seminal vesicle dissection. It is true that the anterior approach does not permit biopsy of the suspicious prostatic nodule.

RETROPUBIC SURGERY IN INFANCY AND CHILDHOOD

We have employed this surgical route to deal with vesical neck and posterior urethral pathology in infancy and childhood. These lesions in the past have been approached either transvesically or transurethrally and neither method in our hands has proved entirely satisfactory. The transvesical approach characteristically lacks adequate visualization and positive hemostasis in vesical neck pathology. The transurethral approach has certain imposed mechanical limitations particularly in infants. We are aware of their apparent satisfactory use in some clinics but our experience has not been entirely without dissatisfaction.

We approach the prostate in precisely the same manner as the adenoma is approached, but instead of using a transverse capsular incision a longitudinal incision is employed which extends from the apex of the gland to the prostatovesical junction. At the superior angle of the incision the lateral branches of the prostatic venous plexus are divided and these vessels are ligated with a single suture on each side of the incision. These sutures are left long to act as tractors and when lateral tension is exerted the entire posterior urethra and vesical neck are adequately exposed for visualization and palpation. We mention palpation particularly since in two instances where the vesical neck obstruction appeared visually to be relieved, we palpated some dense underlying tissue. This was removed and the vesical neck could then be visualized and palpated free of any obstructing tissue.

Following the removal of the tissue about the vesical neck the bleeding points which occur at approximately 5 and 7 o'clock are ligated with a suture of fine catgut and immediately the wound is dry. The prostatic capsule is approximated with a running suture of fine chromic catgut after a catheter of small caliber is introduced into the bladder per urethram. The abdominal wound is closed in layers and a drain is left in place for two days. The catheter is removed on the fourth day and voiding in these children is spontaneous and uncomplicated.

We have now treated 4 such children from three months to nine years of age with excellent results. Where the upper urinary tract was uninfected preoperatively the urine was free of any cellular elements within eighteen days postoperatively. In only one instance, a three month old child, did a suprapubic fistula occur and it closed spontaneously after the catheter was reinserted for a four day period. We have followed 1 of these children for more than a year postoperatively and another for a nine month period; the remaining 2 have been observed six and five months respectively. Where an elevated NPN was stabilized preoperatively by catheter drainage, the level

obtained did not change after operation. Morbidity has been surprisingly insignificant.

SUMMARY

The methods of conservative and radical retropubic prostatectomy are presented. A comparative study of 234 retropubic conservative prostatectomies and 500 perineal prostatectomies is outlined along with a brief discussion of the complications encountered in the retropubic group.

The application of the retropubic method in dealing with various types of vesical outlet obstruction in children is advocated and its application in 4 patients reported.

REFERENCES

Bacon, S. K.: Retropubic prostatectomy, an extravesical technic. J. Urol., 59:376, 1948.

Casper, E. J. and Jacobs, L. C.: A new method of prostatectomy. California & West. Med.., 38:263, 1933.

Farabeuf, L. H.: Les vaisseaux sanguins des organes genitourinaires du perinee et du pelvis. Paris, Masson et Cie., 1905.

Grant, O., Lich, R., Jr. and Maurer, J. E.: Retropubic prostatectomy. Urol. & Cutan. Rev., 52:9, 1948.

Grant, O. and Lich, R., Jr.: Rationale and results in retropubic prostatectomy. Ann. Surg.. 127:1010, 1948.

Jacobs, L. C. and Casper, E. J.: Prevesical prostatectomy. Urol. & Cut. Rev., 37:725, 1933.

Lich, R., Jr., Grant, O. and Maurer, J. E.: Extravesical prostatectomy: a comparison of retropubic and perineal prostatectomy. J. Urol., in press.

Lich, R., Jr., and Maurer, J. E.: Retropubic surgery. Kentucky M. J., March 1949.

Lich, R., Jr., Maurer, J. E. and Burdon, S.: An extravesical method for the relief of vesical occlusion. Arch. Surg., in press.

Lidski, mentioned by Mr. W. K. Irwin (London) in the Scientific Meeting of the British Association of Urological Surgeons June 28, 1946. British J. Urol., 18:118, 1946.

Lowsley, O. S. and Gentile, A.: Retropubic prostatectomy. J. Urol., 59:281, 1948.

Maurer, J. E., Lich, R., Jr. and Burdon, S.: Retropubic vesicolithotomy in preparation.

Millin, T.: Retropubic prostatectomy. Lancet 2:693, 1945.

Millin, T.: Retropubic Urinary Surgery. Baltimore: The Williams and Wilkins Company, 1947.

Millin, T.: Retropubic prostatectomy. J. Urol., 59:267, 1948.

Rosenberg, M. L. and Vest, S. A.: Osteitis pubis. J. Urol., 60:767, 1948.

Tsaknis, D.: Les pedicules vasculaires de la vessie et son peritoine chez l'homme. Arch. d. mal. d. reins, et d'organes genitaux-urinaires, 4:442, 1929.

Van Stockum, W. J.: Prostatectomia Suprapubica extravesicalis. Zentralblatt für Chirurgie, Leipz., 36:41, 1909.

Zuckerkandl, O.: Ueber prostatohypertrophie. Berlin Klin.—therap. Wchnschr., 13:341, 1906.

THE SURGICAL MANAGEMENT OF URINARY TRACT INJURIES*

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SHREVEPORT

Successful management of injured organs of the urinary tract depends upon strict adherence to four important points: (1) the treatment of shock when present; (2) arresting hemorrhage; (3) surgical repair of the injured organ; (4) ample provision for drainage of extravasated urine.

THE KIDNEY

The clinical evidence of renal injury will be one or more of the following: (1) pain in the renal area; (2) shock; (3) hematuria; (4) a palpable mass in the flank. Of these four, pain is the only constant finding and may be either mild or severe. Shock may be absent at first but develops in a matter of a few hours depending upon the amount of blood loss, either through the urinary tract or into the perirenal space. The amount of initial shock also depends upon whether there is associated injury to adjacent organs. Hematuria is variable, if present, and its quantity is no indication of the extent of injury to the kidney.

The kidney may be severely traumatized but no hematuria be present, due to: (1) blockage of the ureter with blood clots; (2) severance of the ureter; (3) detachment of the kidney; or (4) severe vascular injury with renal infarction. A palpable flank mass is evidence of perirenal extravasation of blood, or urine, or both.

In attempting to evaluate the seriousness of renal injury several factors are to be considered: The first is the condition of the patient. Even though the voided urine is grossly bloody and pain is severe, if the

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condition of the patient is good and there is no mass palpable in the flank, conservative treatment is in order. If, however, the patient is in profound shock and is not responding to shock treatment, one should suspect that the renal pedicle is lacerated.

In some cases the history of the injury may be helpful in deciding the extent of the injury. The kidney, being a movable organ, is more likely to escape serious injury if the blow comes from the rear. In this case, the kidney would not be impinged against any immovable organ. However, if the force of the blow comes from in front or from the side, the kidney would be forced against the vertebral column and serious injury would be more likely to result. There are, of course, exceptions to this rule. Excretory urograms (intravenous pyelograms) are at times helpful in determining the extent of the injury. If the dye is well concentrated and confined to the kidney and ureter, extravasation of urine can be ruled out. However, if the dye is seen to be diffused throughout the perirenal area, this is evidence of complete rupture of the kidney or pelvis with extravasation. The excretory urograms will also reveal the status of the opposite kidney, which is essential if removal of the injured organ is considered. When in doubt, cystoscopy and retrograde pyelograms will clinch the diagnosis.

Whether or not surgical intervention is necessary will then depend upon: (1) the condition of the patient, and (2) the extent of the injury. Injuries of the kidney range all the way from mild contusions and lacerations to complete fragmentation of the organ and laceration of the pedicle. If surgery is deemed advisable, it must necessarily be in the nature of an exploration, being prepared to do whatever seems necessary. If associated injury to other abdominal organs is suspected, the transperitoneal approach is in order. If, however, the kidney is apparently the only injured organ, the classical lumbar approach is better.

If clots are present in the perirenal space, they must be evacuated. If the capsule of the kidney is not torn but a hematoma is present underneath, it is best to incise the capsule and liberate the trapped blood in order to prevent future fibrosis with atrophy of the kidney. The kidney has remarkable recuperative powers, and, even though it may be found fractured into several pieces, successful repair can be done by placing pads of fat or some type of hemostatic gauze, such as Oxycel, between the fragments after which the fragments are approximated to something resembling the normal anatomical kidney, using mattress sutures of chromic 1. These sutures are tied over pieces of fat to prevent their cutting through the kidney tissue. Ribbon catgut is preferable, if available. This type of catgut is weaved in and out of the capsule to form a basket-like sling. The ends are tied to approximate the fragments. Tears of the pelvis are lightly closed with chromic 0000. Penrose drains are placed above and below the kidney before the incision is closed.

To summarize, conservative treatment is justified in renal injuries as long as the condition of the patient remains good. If, however, blood pressure and hemoglobin are dropping and the pulse rising, continuous or increasing hemorrhage should be suspected and exploration carried out. Exploration should be done in all cases where there is a palpable flank mass present.

THE URETERS

Ureteral injuries result either from trauma or surgical accident. Ureteral trauma is very rare due to the mobility and the small size of the ureter. The majority of ureteral injuries occur incident to operations on the bladder and female pelvic organs. These are frequently recognized at the time they occur and immediate repair may be accomplished. If the ureter is completely severed, the ends may be sutured over an indwelling catheter. If such catheters are not available, a T tube may be used, one arm of the T extending up and one down the secured ends of the ureter. Often the ureteral injury, when unilateral, remains unobserved, and the kidney dies silently or with insignificant symptoms. The common postoperative indication of open

ureteral injury is a urinary fistula, the urine escaping by way of the vagina or operative incision.

THE BLADDER

Injuries of the bladder should be suspected in all lower abdominal injuries and pelvic fractures. Clinical evidence of bladder injury may be entirely lacking at first, or may be suspected, if the patient has an intense desire to void but is unable to do so, or at most passes a few drops of bloody urine. He should be catheterized, and, if there is doubt as to whether or not the bladder is intact, a measured amount of sterile water should be injected through the catheter and the recovered amount also measured. At least 300 cc. should be injected. If all of the water is recovered, one may safely assume that the bladder is intact. If possible, in cases of doubt, a cystogram should be made by injecting at least 300 cc. (one-fourth strength) of one of the opaque substances used for intravenous pyelograms, such as Skiodan or Diodrast. These substances are nonirritating to the surrounding tissues in case bladder rupture is present. Bladder ruptures may be intraperitoneal or extraperitoneal, and it may be impossible to determine preoperatively which type of rupture is present. If the rupture is intraperitoneal, symptoms of peritonitis do not appear early. In fact, the peritoneum is very tolerant of sterile urine. If the rupture is extraperitoneal, extravasation of urine will take place into the perivesical tissues, but again this will not be clinically evident for many hours.

Early surgical intervention is extremely important in rupture of the bladder. If rupture cannot be proved but is strongly suspected, one is fully justified in exposing the bladder to settle the question. The treatment will depend upon the condition found. The peritoneal cavity should be explored. If the rupture is intraperitoneal, the laceration will be found on the posterior wall of the bladder. The urine is aspirated from the peritoneal cavity, the bladder repaired, using medium size chromic, and the peritoneum closed without drainage, or with a small drain brought out through a stab incision. After the peritoneum is closed the bladder should be opened extraperitoneally and a de Pezzer catheter anchored in the dome of the bladder. Drains are placed down to the base of each side of the bladder before the incision is closed.

If the laceration is found to be extraperitoneal, it is closed and a de Pezzer catheter placed in the dome of the bladder. If the laceration is very small and not easily accessible, it may be ignored, but in all cases the de Pezzer catheter should be placed in the bladder and ample drains placed down to the base of the bladder on each side. The drains should be left in place from five to seven days. The de Pezzer catheter is usually removed in about ten days.

THE URETHRA

Rupture of the urethra should always be suspected in pelvic fractures, injuries of the perineum, and particularly, in straddle injuries, such as falling astride a hard object. The clinical evidence is pain, shock (may be entirely absent), and retention of urine. The patient may pass a few drops of bloody urine on his first effort.

The most common sites of rupture of the urethra are the bulbous and the membranous portions. When urethral injury is suspected the patient should be immediately catheterized. If this is successful, rupture of the urethra can, as a rule, be eliminated. Evidence of extravasation of urine will not be evident at first . If the rupture is in the bulbous urethra, extravasation will take place deep to Colles' fascia, which is continuous with Scarpa's fascia on the abdomen. In due course of time clinical evidence of extravasation will be present throughout the scrotum, and, if allowed to continue, will spread up the abdominal wall.

If the membranous or prostatic urethra is ruptured, extravasation takes place above the urogenital diaphragm in the space of Retzius, but again this will not be clinically evident for many hours.

Early surgical intervention is essential in all cases of rupture of the urethra. Primary repair of the ruptured portion is ideal, but may be impossible to carry out due to the condition of the patient, and in

some cases due to associated pelvic fractures. If primary repair is not possible immediately, cystoscopy is performed plus ample drainage of the areas of extravasated urine. In this case secondary repair of the ruptured urethra should not be delayed longer than absolutely necessary, because dense scar tissue will form at the site of rupture and make further repair extremely difficult.

If primary repair is considered feasible, the approach will depend, of course, on the site of rupture. If the rupture occurs in the bulbous portion, it may be exposed through the perineum and a search made for the torn ends of the urethra. If found, they should be approximated over an indwelling urethral catheter using a few sutures of fine chromic. Ample drains should be placed in all areas of extravasation before the incision is closed.

Ruptures of the membranous and prostatic urethra are not easily accessible, and successful repair may require combined cystotomy and perineal exposure with the patient in the exaggerated lithotomy position. For successful repair each end of the urethra must be located. This may be accomplished by passing a sound through the urethra to the point of rupture and then passing another sound in a retrograde manner through the bladder from above. Then, through the perineal exposure the ends of the two sounds are located, which will denote the site of the rupture. The tissues overlying the two sounds are then incised and the two ends of the urethra located. The sounds are then removed and a catheter is passed through the urethra and guided into the bladder from the perineal side. The urethra is loosely approximated with fine chromic. The perineal incision is closed with ample drainage, after which a de Pezzer catheter is placed in the bladder before it is closed, with ample provision being made for drainage of the perivesical spaces.

In conclusion, one should remember that early surgical intervention is essential in all injuries of the lower urinary tract where rupture of the bladder or urethra is proven or strongly suspected. However, in injuries of the kidney, early surgical intervention is the exception rather than the rule, and one should adopt a conservative attitude as long as the patient's general condition remains good.

DIFFICULT LABOR IN RELATION TO FORCEPS DELIVERY*

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The management of dystocia, or difficult labor, continues to be one of the most important phases of obstetrics, in spite of prognostic refinements of pelvimetry and the increased safety of cesarean section.

Because of this, we felt that a critical analysis of our most difficult deliveries throughout the years would contribute lessons of service and bring out principles of value in the future management of such deliveries, for dystocia problems will surely always be encountered. Consequently, we reviewed the records of about 3,000 operative deliveries, occurring during the years of 1932 through 1948, on the obstetric service of the Section on Obstetrics and Gynecology of the Mayo Clinic. In this period, about 12,500 deliveries occurred. The records of 5 patients who had extremely difficult deliveries prior to 1932 came to light in the survey and are included. A total of 86 cases was selected, although we are sure some very difficult deliveries were overlooked. The incidence of extremely difficult deliveries, therefore, was 0.69 per cent or about 1 for each 145 deliveries.

It should be emphasized that these cases represent the most difficult deliveries through the pelvis in a sizeable group of obstetric patients over a seventeen-year

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period. Many were admitted in labor with or without prior attempts at delivery and with no opportunity for prenatal study and planning for the best type of delivery. We seemed committed in these cases to operative delivery from below. In many other cases, in spite of prenatal care in our own group, we found "judgment difficult and experience fallacious" so that honest errors in judgment gave us these very hard forceps deliveries. The experience presented is that of our own group and we know the results are correct even though not always flattering.

The records of the cases were studied and the facts will be presented in the following sequence: parity, type of delivery, type of pelvis, quality of uterine contractions, length of labor and weight of the fetus. We then studied the incidence and nature of subsequent deliveries and finally the maternal and fetal results. We were especially interested in the remote results of these difficult deliveries on the fetus.

PARITY

For 66 patients, it was the first delivery and for 16 the second delivery. The remaining 4 patients were multiparas, having had 2 to 4 previous deliveries. In each instance of multigravity but 2, the babies were decidedly larger than those previously delivered, this fact causing the dystocia.

TYPE OF DELIVERY

Except for 1 craniotomy and 3 breech deliveries wherein unusual difficulty was encountered in the application of forceps to the after-coming head, all deliveries were by forceps with the vertex presenting. There were 18 low forceps deliveries, 60 midforceps deliveries and 4 high forceps deliveries.

The 4 cases of high forceps delivery require some comment. We subscribe emphatically and heartily to the idea that the employment of the high forceps operation is, in general, poor obstetric practice. On the other hand, we feel that there are exceedingly rare exigencies wherein the employment of the high forceps procedure is justified or even indicated. With 4 instances of this type of delivery in 12,500 deliveries (1 in 3,125 patients), it is seen that its use was very infrequent. Two of the 4 high forceps deliveries occurred in 1932, the first year included in this study. In 1 instance, the baby was dead at the outset and high forceps delivery seemed to the physician to be a less traumatic procedure than did craniotomy on a high head in a patient with severe pre-eclampsia complicated by pulmonary edema.

In the remaining 3 cases, we feel that high forceps delivery gave the infant its only chance of survival, and 2 of the 3 infants did survive without injury, 1 of the 3 being stillborn. The following mention of 1 of the 4 cases may exemplify the uncommon indication for high forceps delivery.

A 23 year old woman, gravida 2, para 1, was admitted in 1945 with profuse hemorrhage from a marginal placenta previa. She was given 2,000 cc. of blood and the hemorrhage was controlled by Voorhees' bag. When the bag was expelled the placenta was about four-fifths detached and in the vagina, yet the heart tones were audible, though indistinct and irregular. The head was high in the pelvis and could not be engaged with fundal pressure, though the baby was small. The uterus was dry and there was not time enough to secure the desired relaxation by anesthesia to perform a version and extraction. The occiput lay in the right occipito-transverse position; high forceps were readily applied, and rotation and descent easily accomplished. The placenta came over the perineum ahead of the fetal occiput, but the fetus, weighing only 2,710 gm., responded in five minutes to the usual treatment for asphyxia and the child is now 3 years old, alive and well.

In another instance of a primipara, the cord prolapsed just as the cervix was almost dilated with the head of an average-sized fetus at a station slightly above the ischial spines. A relatively easy application of high forceps resulted in the delivery of a normal infant but this woman suffered from the only ruptured uterus in this series, in spite of the fact that the application of the forceps and the traction required did not seem to the physician to be excessively traumatic. This case points out the gravity of high forceps delivery, and on the rare occasions when it is used, the physician must realize the grave responsibility he undertakes.

TYPE OF PELVIS

A surprising fact appears when the pelvic measurements, determined by the usual office methods or by more exact modern methods of roentgenologic pelvimetry or by both, are studied. Forty, or nearly one half, of the patients had apparently normal pelves. This number is perhaps too high as many were seen before present-day accurate pelvimetric methods were available. Yet, we believe that many of the 40 did have what could be termed adequate pelves because, first, they were found to have adequate pelves on recent remeasurement or, secondly, they had delivered fetuses of excessive size. Twenty-five women of the group had some definite degree of pelvic contraction. Twenty-one had quite severe degrees of pelvic contraction.

No doubt many of the patients with so-called normal pelves had pelves that were inadequate for the large fetuses they bore. That is, in spite of normal pelvic measurements there was cephalopelvic disproportion. In 9 of the patients with normal pelves the dystocia occurred as the result of other factors than the babies' size. Such factors were locked twins in 1 case, prolapsed cord, placenta previa, hydrocephalus and so forth. In the remaining 31 cases, however, wherein the pelvis was normal, the *average* fetal weight was 4,000 gm. More will be mentioned later as to the importance of fetal weight.

QUALITY OF UTERINE CONTRACTIONS

The uterine contractions were unsatisfactory to the extent that they were fair or poor in quality in exactly half of the cases. This was not an unexpected finding but bears out the impression that uterine inertia is a disturbing and annoying accompaniment in cases of dystocia. The obstetrician has cause to yearn for good uterine dynamics when his dystocia suspect goes into labor.

LENGTH OF LABOR

As would be expected, the length of the first stage was often prolonged. Forty-two, or nearly one half (48.8 per cent) had labors that lasted twenty hours or more and 22, or one quarter, had first stages of labor that lasted thirty hours or more. The average duration of the first stage for the group was 24.8 hours.

There was a considerable variation as to the time that patients were allowed to remain in the second stage. For example, 31 patients were delivered by forceps operation before the cervix had been completely dilated one hour. Nine patients who had Dührssen's incisions are included in this group, as well as some of the multiparous patients. Maternal and fetal exhaustion accounted, also, for rather early interference after complete dilatation had been attained in some cases. Ten patients were allowed to remain in the second stage for four to seven hours. Two patients had a second stage of more than ten hours; in 1 of these, there was uterine inertia.

WEIGHT OF FETUS

The fetal weight was the only accurate measurement on the records that seemed indicative of fetal size, and we were impressed with the importance of fetal weight in these examples of extreme dystocia. Sixty-five, or 75.6 per cent, of the babies were above the usual yearly average in fetal weight (3,325 gm.) of clinic patients; the average weight of the 86 babies was 3,714 gm. Twenty-eight, or one third, of the babies, weighed more than 4,000 gm.

SUBSEQUENT DELIVERIES

The future of the 86 mothers following their experience of extreme dystocia is of much interest. They remained unusually infertile, as is evidenced by the fact that apparently only 33 of the 86 women went through a subsequent pregnancy. These 33 women had a total of only 45 babies subsequent to their experience of the original hard labors and deliveries. From examination of the case records, we do not believe that there were many instances in which they sustained enough morbidity to render them sterile, but rather the infertility was voluntary. Either the patient or her husband, or both, did not wish to risk a similar ordeal again. While this attitude is understandable, it may not be logical because those who did venture another pregnancy were delivered of normal babies with

relative ease, except for 3 repeated mid-forceps deliveries. So far as we can ascertain, there was no fetal mortality or morbidity in the 45 deliveries following the dystocia.

The fetal weight is again of interest in these subsequent deliveries. In 20 of 27 cases in which records were available and in which the delivery was through the pelvis, the weight was less than that of the infant presenting the original dystocia. In 3 of the 7 exceptions other obstetric difficulties explained the discrepancy. The prior clinical impression of one of us (A.B.H.) that often a patient having dystocia as a primipara will deliver a heavier baby with ease (the primary dystocia being attributed to uterine inertia or "soft part dystocia" rather than to real disproportion) is largely refuted by this study. The weight of the fetus in subsequent deliveries was frequently 500 to 1,300 gm. less than in previous deliveries. The patients undergoing cesarean section in pregnancies following their dystocia had, in 9 instances, fetuses whose average weight was 4,014 gm.

MATERNAL RESULTS

As inevitably happens in such a series of difficult operative deliveries, there was considerable trauma and loss of blood. The blood lost was normal (less than 500 cc.) in 43 cases. In 10 cases, the loss of blood was excessive, but transfusion was not required. Thirteen patients either sustained enough hemorrhage (estimates of 500 to 2,000 cc.) to require transfusion of blood or went into shock without hemorrhage. Six patients received 1,000 cc. or more of blood. A ruptured uterus accounted for the hemorrhage in 1 case and placenta previa augmented the loss of blood in 2 others. There was no maternal death in the 86 cases.

IMMEDIATE FETAL RESULTS

There were 8 stillbirths and 4 neonatal deaths in the 86 deliveries, giving an uncorrected fetal mortality rate of 14 per cent. One infant was macerated and a high forceps delivery, even though the infant was dead, seemed preferable to a craniotomy on a high head, since the mother was in precarious condition with severe pre-eclampsia and pulmonary edema. Congenital anomalies incompatible with life caused the dystocia in 2 cases. Another patient was admitted with a large fetus and delivered save for the head, requiring a craniotomy on the after-coming head. In a fifth case, a marginal placenta previa probably contributed to a stillbirth of a 5,400 gm. fetus. The corrected infant mortality was, then, 7 infant deaths, or a rate of 8.1 per cent, in which operative trauma destroyed the fetus. Many babies had various evidences of delivery trauma on the head, such as forceps bruises, cephalohematomas, and transient facial paralysis. Two infants had external eye injuries from forceps, 1 requiring suture of the eyelid, but with quite satisfactory late results.

LATE FETAL RESULTS

The remote fetal results of these difficult deliveries were of special interest to us. Twenty-one of the 74 infants who survived were dismissed from the hospital at the end of the neonatal period or slightly before (average of about eleven days) and have never returned to the clinic. This is, admittedly, an unsatisfactory follow-up. All were neurologically and otherwise apparently normal, although 1 infant had had one jacksonian convulsion soon after birth. The records of 20 of these babies were examined to ascertain these results.

In 10 other instances, the mothers' records indicated that the babies went home in good condition, but no follow-up data were available. This left 43 infants who have been followed-up for six weeks to twenty-six years. Of these 43 babies, 5 were followed up for one year and 17 others for five years; in these 22 children there is, of course, still the possibility that difficulties due to their hard deliveries may yet appear. It was in this group that the 1 impaired child appeared, with epilepsy attributed by the neuropediatrician to birth injury. Fifteen of the 43 children have been followed up for ten to twenty-six years and all are apparently normal, save 1 who died at the age of ten years of melanosarcoma of the neck. One child, at the age of 1 year, had

partial inability to rotate externally the right lower extremity. The cause of this was indeterminate on examination by the orthopedist who instructed the parents to return with the child if the difficulty persisted. This they have not done.

In summary, 43 children have been followed up for six weeks to twenty-six years with only 1 having gross evidence and 1 having questionable evidence of neurologic or other birth injury.

COMMENT

This study has forcefully re-emphasized for us the fact that difficult labor involves the three factors of pelvic capacity, fetal size and uterine dynamics. In other words, this is the old passage, passenger and powers triad that DeLee and others have always stressed. There is often a fourth factor of the soft parts, the resistant cervix or perineum, the vagina and its supports or adipose tissue in the obese, but these factors are harder to exaluate in studying patients' records in retrospect.

While we have come to have the greatest respect and admiration for the pelvimetric prognostications of the roentgenologists and owe a great debt to their services, the roentgenologists readily agree with us as to the incompleteness of their prognosis, owing to the other factors of fetal size and uterine performance. In any event, it is clear that the management of dystocia cannot be statistically carried out by the obstetrician from a swivel chair with the pelvimetry films before him in a view box. He must repeatedly and patiently study the frequency, regularity, and intensity of the uterine contractions and make himself as expert as he can in the most difficult art of estimating the size of the unborn infant. He must perfect himself in the technic of determining rectally or vaginally the station of the presenting part and appreciate by abdominal examination also the degree of engagement or the lack of engagement. Finally, with these facts and by fine judgment and experience, he must settle, before the best time for cesarean section is gone, whether or not it is reasonable to try a delivery through the pelvis. Almost never is it necessary in modern obstetrics for a woman to lose a baby in order to determine what type of delivery is best for her. To do so twice is almost inexcusable.

Mussey and Hunt, and a decade later, DeVoe and Hunt, studied the subsequent deliveries of women who had lost 1 or more babies from dystocia. Only 1 of 64 such women was unable in subsequent deliveries to have 1 or more normal children and this patient had a severe hypertension resulting in repeated stillbirths or macerated fetuses.

The high incidence of essentially normal pelves in this series of 86 difficult vaginal deliveries may be questioned. Some of the earlier patients still remain to be subjected to more accurate present-day pelvimetry although it should be mentioned that in many this study has been done. In some, degrees of contraction were detected while in others the normal capacity was confirmed. If the fetal size is excessive, there exists an absolute or relative disproportion even in the normal pelvis. This is aggravated if the uterine contractions are of poor quality. The figures related above show the importance of fetal size as expressed in terms of the weight of the fetus. The weight of the fetus is an admittedly poor method of determining the size and capacity of the fetal head, but it is perhaps the best measurement we have for it today. Our records of fetal weight were complete, and we feel accurate in each case.

Twenty years ago, no one was satisfied with the accuracy of pelvimetry. Possibly within twenty years, we shall have a practical, simple, and accurate type of cephalometry, roentgenologic or otherwise, but nearly everyone — obstetrician and roentgenologist alike—agrees that such is not now available. It would be a most welcome adjunct to the management of dystocia.

After we noted the importance of fetal weight in this study, we reviewed the records of 116 cases of tests of labor, 55 of which were unsuccessful and 61 of which were successful. Among the successful tests there were only 4 infants (6.6 per cent) whose weight exceeded 4,000 gm. *and 3 of these 4 cases already had been encountered*

in our group of 86 cases of dystocia! The fourth patient delivered spontaneously an infant weighing barely more than 4,000 gm. In the 55 unsuccessful tests, there were 16 infants (29.1 per cent) whose weight exceeded 4,000 gm. Thus the incidence of large babies was four and one-half times as high in the group in which tests of labor failed and cesarean section was required as in the group of patients who succeeded in their tests of pelvic delivery.

Obviously, many women with ample pelves can and do deliver large babies without undue difficulty, but the importance of fetal size looms large in our group of women who required hard forceps deliveries even though the pelvis seemed ample in many instances. When a patient with a large fetus does poorly in labor, cesarean section should be contemplated though the pelvis seems adequate. It matters little whether the pelvis is classed as "normal" if it is too small for a large fetal head or if cephalopelvic disproportion exists. Naturally, such indications will not often arise and the conscience of the physician is involved just as for any other indication for cesarean section.

The fact that exactly half of the patients in this dystocia group had unsatisfactory uterine contractions, which were classified as poor or fair rather than good, speaks for itself as to the importance of this function as one of the triad of dystocia.

When one recalls that the cases were selected because of the difficulty of the delivery, over a period of seventeen years, a high fetal mortality could be expected and did occur. The remote results were extremely encouraging in that only 2 of 43 children who had been followed up for months and years showed any question of neurologic injury. No injury is known among the remaining 31 infants; they were all dismissed from the hospital in satisfactory condition. The population from which our obstetric material comes is a fairly settled one and most of their medical care is received at the clinic; thus it is reasonable to believe that we would have been acquainted with the majority of troubles that might have arisen in the group of children. Some few instances of difficulty may well have occurred, of course, among infants whose parents moved away or sought care elsewhere.

We conclude, then, that if the infant survives the delivery and the neonatal period, its chances for a normal life are excellent. This most encouraging result was not entirely unexpected, since Keith and Norval are reporting similar results in the independent pediatric study of some of our more recent obstetric material. They show little if any increase in neurologic difficulties in children born after prolonged labor or in infants suffering fro masyhyxia neonatorum as compared with control groups, although the initial risk was likewise higher from these factors.

The infertility of these patients following their hard deliveries is cause for concern. At present we believe that this is on a voluntary basis. Only 40 per cent, to our knowledge, have since become pregnant. Here, too, the figure of which we have exact knowledge may be lower than the actual figure of subsequent reproduction, but it seems certain that fertility is low when there have been only 45 babies born to these 86 women since their difficult deliveries. Here is a responsibility for the obstetrician. We agree with the attitude of Cosgrove that the woman who has suffered the ordeal of an unusually difficult delivery should virtually be promised that such an experience will not be duplicated. In general, this can be almost universally effected and contrary to some opinions, without an excessive resort to cesarean section. The incidence of cesarean section in our patients who later became pregnant again was 18.2 per cent, which is not a very high figure when one recalls how this group of cases was selected. There was no fetal mortality in the subsequent pregnancies, nor findings of fetal injury. However, probably 3 of the 27 patients delivered from below might have had abdominal delivery, as they involved a repeated midforceps delivery. If such patients are subjected to a test of labor it should not be too rigorous. We sug-

gest that after an especially difficult delivery, the attending physician note his recommendations for the management of subsequent deliveries while salient points of his difficulties are fresh in his memory. These suggestions may not always appear sound later, but will at least warn others who may in the future be responsible for attendance on the woman.

CONCLUSIONS

1. Pelvimetry alone, while of great value as a prognostic guide in dystocia, is not enough. The fetal size as measured by its weight is of tremendous importance as is study of the quality of the uterine contractions.

2. The immediate fetal mortality rate in our series of cases was high; the uncorrected rate was 14 per cent, and the corrected rate about 8 per cent. However, the remote results as to the surviving children have been very encouraging. There was no maternal death.

3. The infertility following the type of delivery studied is disconcertingly high. The physician should be able to increase the fertility rate subsequent to severe dystocia by a discussion with the patient and her husband. He should be able to promise that there will be no repetition of a grueling ordeal and yet offer a good fetal prognosis. With care and planning, this promise can almost invariably be kept.

DISCUSSION

Dr. E. L. King (New Orleans): First of all, does Dr. Hunt agree with the idea that textbooks should speak more of disproportion, rather than of contracted pelvis? I think that it is important that we should emphasize the question of disproportion. Secondly, does he not agree that the radiologists' reports should give the size of the pelvis and fetal head without expressing an opinion as to whether the baby will get through or not?

Dr. Arthur B. Hunt (Rochester, Minn.): I heartily agree with Dr. King's points. The crux of the matter is summarized in the word, disproportion, and it is rather immaterial whether the pelvis is normal or not if the baby is too large for it.

Secondly, the radiologist probably should report what he sees and let the obstetrician determine the prognosis in labor from the pelvimetry films. One of our radiologists is expert in offering a prognosis, but even so, we care more for his general opinion about the pelvis and its measurements, exactly because he can not estimate the quality of the pains and determine exactly the size of the fetus.

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SIGNIFICANCE OF THE FETAL HEART RATE IN PREGNANCY AND LABOR

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The frequent auscultation of the fetal heart tones is common practice on all obstetrical services. Their significance is not usually appreciated except as a prognostic sign in regard to the status of the fetus. It is our purpose here to discuss the significance of the fetal heart tones, the mechanism by which changes in rate occur, and the management of cases in which these changes in rate and rhythm are present.

HISTORICAL DATA

The fetal heart tones were first heard in 1818 by F. I. Mayer, a surgeon from Geneva. In 1821, Lejumeau de Kergaradec, an obstetrician and associate of Laennec, also heart the fetal heart tones and uterine souffle; he showed that the maternal pulse had no effect on the fetal heart beat. Naegle, in 1850, found that the fetal heart rate was decreased by compression on the cranium of the baby at the time of its passage through a narrow pelvis. One of the first to advise immediate delivery of a fetus whose heart rate exceeded 160 per minute was Winckel in 1903. In 1925, Bartholomew was one of the first to disagree with this management; he felt that rapid rates were not serious, and moreover, infrequently observed. DeLee in 1915 in collaboration with Hillis, devised the head stethoscope which we use today.

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NORMAL VARIATIONS

Fetal heart tones are usually first heard at eighteen weeks' gestation, but may be heard as early as fourteen weeks to sixteen weeks. At the onset, the rate is rapid—around 150 per minute and most frequently heard in the midline just above the bladder.

Clinical significance in early pregnancy. The presence of fetal heart tones is a definite sign of pregnancy. It is one of the few physiologic phenomena of the fetus which may be observed accurately and at will. It may be clinically significant by its absence along with the finding of an enlarged uterus, indicating such possibilities as: (1) dead fetus, (2) hydatiform mole, (3) multiple pregnancies, (4) uterine tumors, and (5) spurious pregnancy.

Clinical significance in late pregnancy. The fetal heart rate has a normal variation in late pregnancy—120 to 170 per minute. The rate may be increased by fetal movements and activity, or manipulations of the fetal head by abdominal palpation. Vaginal or rectal examination may cause either an increase or decrease in rate.

One of the earliest manifestations of fetal distress in the form of anoxia may be a change in the fetal cardiac rate. On the other hand, Sontag and Newberry[1] found that three-fourths of all patients have a fetal heart rate of 160 or over, without evidence of fetal distress, at some time during the last two months of pregnancy.

In the last months of pregnancy although the location of the fetal heart tones will often hint at the position of the baby, this finding is not by any means definite as to presentation or position.

Frequently it may become necessary to differentiate between the maternal pulse rate and the fetal heart rate when the former is rapid. To do so, Berlind[2] has used a technic based on the phenomenon of respiratory arrhythmia. The normal maternal beat is accelerated during inspiration due to the decrease of vagus activity and slowed during expiration because of an increase of vagus activity.

The failure to detect fetal heart tones does not necessarily indicate the death of the fetus. Such factors as obesity, a loud maternal pulse obscuring the fetal heart beat and polyhydramnios may be present, making auscultation of the fetal heart tones impossible.

NORMAL VARIATIONS DURING LABOR

During labor, the same variations as noted above in late pregnancy may prevail. In addition, there may be slowing of the rate with uterine contractions or deep sedation. Vasomotor stimulants such as pitocin, or adrenalin (used in saddle block anesthesia) may cause an increase in the rate. If, following spinal anesthesia, there is a temporary drop in maternal blood pressure, changes in fetal heart rate are often observed.

ABNORMAL VARIATIONS DURING LABOR

1. *Rapid rate*: In a survey of the literature, the impression obtained was that transient fetal tachycardia does not necessarily indicate fetal distress. King[3] states that rates up to 170-180 are no cause for alarm providing the uterine contractions are not unduly strong or too close together. He feels that a rapid rate may mean threatened or early asphyxia. Lund[4] has observed that persistent, marked fetal tachycardia is a fallible sign of fetal distress. He has classified fetal tachycardia as either transient or persistent with a time limit of twenty minutes between the two classes. He lists the etiologic factors in transient fetal tachycardia as follows: (a) fetal movements and activity, (b) application of forceps, (c) pressure of the head on the perineum, (d) uterine contraction, and (e) rectal or vaginal examinations. Lund has concluded from his study that transient, rapid fetal heart rates were neither common nor hazardous and that dangerous and unjustified obstetrical intervention was largely responsible for the unfortunate results commonly and erroneously attributed to fetal tachycardia.

On the other hand, Richardson[5] has pointed out that a rapid rate of the fetal heart may be the earliest indication of abruptio placenta and often occurs before evidence of hemorrhage appears. He offers

the explanation that any separation of the placenta alters the oxygen and carbon dioxide exchange between the placenta and placental site; (slight crescentric separations at the periphery usually do not effect the fetal heart rate). As placental detachment increases, the oxygen-carbon dioxide exchange is altered so as to produce fetal heart embarassment to which the fetus responds with compensatory acceleration of the fetal heart rate. The fetal circulation speeds up in direct relation to the ever diminishing available area of placental attachment; this continues until the functioning area becomes so small that it is no longer capable of maintaining the oxygen-carbon dioxide balance. There develops an oxygen deficit; carbon dioxide becomes overwhelming; asphyxia occurs, and the fetal heart rate finally slows down.

2. *Slow rate.* This is the sign of most importance and usually is more grave in significance than a rapid rate. It generally indicates fetal distress when persistent. Some interference with fetal circulation is strongly suggested such as a short cord, a true knot in the cord, a cord around the neck with some tension, or a prolapse of the cord. It must be pointed out that the same conditions that cause a rapid rate may also produce bradycardia.

3. *Irregular heart rate.* King[3] states that the significance of this sign is dependent on the relation of the irregularity to uterine contractions. When noted during contractions, an irregular rate is not significant. If the irregularity lasts for one-third to one-half of the interval between contractions, it generally indicates a coil of cord around the neck with some tension. If the irregularity persists throughout the entire interval between contractions, the baby is in serious danger.

PHYSIOLOGY

The fetal heart rate is influenced by (a) vagus nerve stimulation, (b) the amount of work the heart has to do in transmitting a varying column of blood, and (c) the oxygen-carbon dioxide content of the blood[6]. Anoxia affects the fetus quickly. Clinically, this may be detected by changes in the rate of the fetal heart. The rate first increases, then slows down. The slowing may be due to stimulation of the vagus center by anoxemia, an increase in the carbon dioxide content of the blood by metabolic products of asphyxiation, or by compression of the brain. Continued stimulation of the vagus center results finally in its paralysis, and the pulse increases because of the lack of vagal inhibitory action.[7] Therefore, any condition which interferes with the oxygen carrying system of the fetus or which causes stimulation of the vagus impulses will be manifest by a change in the fetal heart rate. If these conditions are transient, fetal asphyxia will not be a problem. If, on the other hand, they persist, the result will be distress of the fetus. This can be relieved only by removal of the cause or delivery of the fetus.

MANAGEMENT

In no case should operative delivery by the vaginal route be attempted unless the accepted conditions for such a procedure are present.

Management in the first stage of labor, delivery not feasible: If the heart tones indicate progressive fetal distress, cesarean section should be seriously considered. Additional factors may be present to support the choice for this procedure, such as an elderly primipara, cephalopelvic disproportion, or a rigid cervix. The use of oxytoxics should be restricted. Uterine irritability should be treated with sedatives or anesthesia, if necessary. If there is evidence of fetal oxygen lack, sedation should be restricted. The maternal fluid balance should at all times be maintained. Oxygen, given to the mother in the presence of a slow fetal heart rate as a result of fetal oxygen lack, will increase the rate. These cases should be watched carefully with frequent auscultation of the fetal heart tones; the patient should be examined carefully for an occult or true prolapse of the cord. Abruptio placenta should be ruled out.

Management late in the first stage, delivery feasible: One of two procedures may be chosen. Immediate delivery may be accomplished either by Duhrrsen's incisions,

if the cervix is not completely dilated and fails to dilate rapidly; or the patient can be urged to use her contractions if the conditions are such that this will produce delivery in a short period of time. Oxygen should be administered until anesthesia is started. Saddle block anesthesia should not be used here.

Management in the second stage, delivery feasible: Immediate delivery by the least traumatic means should be carried out. In a multipara in whom there is no cephalopelvic disproportion and the head is still high, above a safe plane for forceps, version and extraction is indicated. In breech presentation, immediate extraction should be done.

In any case in which delivery is done for fetal distress, prepare for prompt and efficient resuscitation.

CONCLUSIONS

1. Fetal heart tones are an important diagnostic sign in early pregnancy.

2. In late pregnancy and during labor, a fetal heart rate over 170 does not necessary indicate fetal distress, but may be the earliest sign of abruptio placenta.

3. A fetal heart rate below 100 is usually indicative of fetal distress. It is very unwise to wait for the rate to drop to 100 before interfering.

4. Close observation and general supportive measures are necessary when the fetus is in distress.

5. In no case should operative vaginal delivery be carried out unless the accepted conditions for such procedures are present.

6. Prepare for prompt and efficient resuscitation of the infant in those patients for whom delivery is done for fetal distress.

REFERENCES

1. Sontag, L. W., and Newberry, H.: Am. J. Obst. & Gynec. 40:449, 1940.
2. Berlind, M.: Am. J. Obst. & Gynec. 49:703, 1945.
3. King, E. L.: Am. J. Obst. & Gynec. 39:529, 1940.
4. Lund, C. J.: Am. J. Obst. & Gynec. 45:636, 1943.
5. Richardson, G. C.: Am. J. Obst. & Gynec. 32:429, 1936.
6. Freed, F. C.: Am. J. Obst. & Gynec. 14:659, 1927.
7. DeLee, J. B., and Greenhill, J. P.: Principles and Practice of Obstetrics, 6th ed., Philadelphia, 1933, W. B. Saunders Co., p. 866.

THE IMPORTANT COMPLICATIONS OF CATARACT SURGERY

(EXCLUSIVE OF SYMPATHETIC OPHTHALMIA)

WILLIAM B. CLARK, M. D.

NEW ORLEANS

Spaeth[1] was very near the truth when he said that the complications possible after cataract extraction are greater numerically than those which can follow any other type of surgery, regardless of the anatomic area in which the operation is performed. He might properly have gone further. He might have emphasized the small distance which lies between success and failure in cataract extraction and might have pointed out how often an apparently trivial act or circumstance could turn the scale against the patient. Cataract extraction is not a procedure in which life or death hangs on the outcome. On the other hand, when one considers the social, financial, emotional, and psychic consequences which may follow failure, it behooves the ophthalmic surgeon to omit no single precaution which might contribute to a successful outcome.

While I do not intend to discuss the complications of cataract surgery in relation to special techniques, it might be well to mention the interesting studies on senile cataract published from the Wilmer Institute by Hughes and Owens,[2] one of which has to do with the relation of techniques to results.

In the 2,086 cataract extractions performed between 1925 and 1945, which make up this series, five techniques were employed. Loss of vitreous occurred most often in combined intracapsular extractions and least often in round-pupil intracapsular extractions. The incidence of prolapse of the iris was greatly reduced when corneoscleral sutures were employed, as was the number of cases of delayed wound healing. Anterior chamber hemorrhages occurred most frequently after combined intracapsular extractions performed with conjunctival sutures and least frequently after round-pupil

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intracapsular extractions performed with two corneoscleral sutures. Detachment of the retina was somewhat more frequent when intracapsular extractions were performed with conjunctival sutures. Postoperative iridocyclitis decreased markedly in incidence when total extraction of the lens was practised, and secondary glaucoma became less frequent when intracapsular extraction was performed with corneoscleral sutures. In general, my own results are in agreement with these.

Perhaps the most significant thing about Hughes and Owens' report is their statement that over the nineteen year period, which it covers, the incidence of postoperative complications has become progressively lower while final visual results have become progressively better. The development of new techniques has unquestionably played a large part in that improvement, as has the introduction of chemotherapeutic and antibiotic agents. Even more important, however, has been the general refinement of ophthalmic techniques as distinguished from the introduction of new operative procedures—and the ophthalmic surgeon's increasingly broad concept of his specialty. The improvement which Hughes and Owens report, with the passage of years, is a universal experience. It is particularly striking, as would be expected, in such an organization as the Wilmer Clinic, but it is notable elsewhere, in other organizations and in the experience of individual practitioners.

ACCIDENTS AT OPERATION

The complications of cataract surgery begin on the operating table, though at this time they might more properly be called accidents, or, in some unfortunate instances, catastrophes. On most of them I shall spend no great amount of time, but they are worth discussing briefly, for some of them can be prevented and others, if they cannot be prevented, can be controlled, or at least minimized, when they have occurred. Prolapse of the iris, and intraocular hemorrhage, which may occur both at and after operation, as a matter of convenience are discussed under postoperative complications.

Movement of the eye before the completion of section may jerk the knife out of the eye or may cause the section of the cornea to be completed at a more or less oblique angle. In the former event, if no damage has occurred, a conjunctival flap is prepared and the section is completed by means of sharp scissors with blunt tips. If the incision has been completed at an undesirable angle, nothing can be done except to proceed with the operation; sometimes it may be wise to use a sliding flap over the corneal incision.

If *orbicularis spasm* occurs, it must be relaxed as promptly as possible because it predisposes to vitreous prolapse. The patient is told exactly what has occurred, without, of course, alarming him, and is instructed to open the opposite eye. Meantime, while the pull on the affected lid is slightly relaxed, the eyebrow is lifted and is immobilized with digital pressure at the rim of the bony orbit. This simple maneuver usually controls the action of the occipitofrontalis muscle.

Expulsive extrusion of the lens, or dislocation of the lens, may occur as the corneal section is accomplished and is occasionally so precipitate that the lens actually shoots from the eye. This is an extremely serious accident because the amount of vitreous lost at the same time is usually so great that phthisis bulbi is almost inevitable, even if immediate subretinal or choroidal hemorrhage does not follow.

Rupture of the capsule during delivery makes complete removal of the lens material more difficult. It is essential that this material be removed, for it predisposes to postoperative uveitis, as well as to glaucoma and may require such secondary procedures as discission or capsulectomy, which are not simple operations. When the capsule ruptures, the method of extraction depends upon the position of the lens when the accident happens. These are matters of technique, which I shall not take time to discuss except for the warning that if there is any pressure on the eye, with the asso-

ciated possibility of vitreous loss, the residual capsule had best be left alone after the lens material has been removed.

In the occasional case, especially when the lens is hypermature, it will be found that the moderate trauma associated with corneal section has resulted in *rupture of the zonula*, though the vitreous remains intact. It is sometimes possible to extract the lens with a smooth capsule forceps under these circumstances, but more often the loop must be used. If the vitreous presents before the extraction has begun, one has no choice but to use the loop, since in such cases the hyaloid membrane has ruptured as well as the zonula.

If *fragmentation of the nucleus* occurs during operation, each individual fragment must be removed with smooth forceps or by irrigation. Retention of the nucleus and of any great amount of cortex is highly dangerous, because it may lead, as will be pointed out later, to serious iridocyclitis or ophthalmitis phaco-anaphalactogenica, which may be followed by the loss of the eye. If the case is also complicated by presentation of the vitreous, removal of the nucleus and cortex furnishes real problems, since every manipulation increases the loss of vitreous.

The *loss of vitreous* which is strictly fluid (that is, unformed) is not a matter of any consequence if it is not excessive, since the anterior chamber will re-form as soon as the lens is delivered. The loss of formed vitreous, however, will inevitably occur if more than a certain amount of fluid vitreous is lost. It is particularly distressing when it occurs after the cataract has been extracted and the essential part of the operation is over. Loss of any significant amount causes vitreous opacities, renders the preservation of a round pupil impossible, and predisposes to the development of inflammation, glaucoma, late detachment of the retina, and haziness of the cornea.

When prolapse of formed vitreous occurs, the bridle sutures are immediately released and the patient is told to look upward, or, if anesthesia does not permit his cooperation, the upper lid is lifted away from the eyeball, the lid elevator is removed and the lids are closed. After a minute or two, the lid elevator is again introduced and the conjunctival sutures are tied with the eyeball in upward rotation. The eyeball is then depressed sufficiently to permit the introduction of the iris spatula and the vitreous which lies between the wound edges is resected. Operative maneuvers must be completed as promptly as possible. As Spaeth[1] says, attention to this simple detail has probably saved more eyes than any number of fussy, complex manipulations.

Loss of vitreous seldom occurs if there is no pressure on the eye. Pressure can be prevented, as Guyton[3] points out, by akinesia of the orbicularis and the extraocular muscles, an adequate palpebral fissure, secured by canthotomy if necessary, and lack of excessive retrobulbar bulk. General anesthesia should be used in patients who present the latter finding, or in whom there is excessive intraorbital resistance, since retrobulbar injection will aggravate the situation.

POSTOPERATIVE COMPLICATIONS

Prolapse of the Iris. Prolapse (or retroversion) of the iris which occurs at operation, before extraction, is usually impossible to correct. Peripheral iridotomy sometimes takes care of the situation, but as a rule complete iridectomy is necessary.

Partial or complete iriodialysis may occur in the course of iridectomy, either because the patient moves his eye while the iris is in the grasp of the forceps, or because the surgeon removes the forceps with the iris in its grasp, without completely sectioning it with iridectomy scissors. If iridodialysis is partial, as is likely, it may be possible to stroke the iris back into the angle in such a manner that the complication will not be apparent after healing has occurred. If iridodialysis is complete, the only possible remedy is pinhole glasses.

Perhaps the most common cause of postoperative prolapse of the iris is difficulty in repositing the iris at operation, because of bulging of vitreous into the wound. Loss of vitreous, however, is not a predisposing factor. The incidence of prolapse of the

iris has been greatly reduced by the use of corneoscleral sutures, and when this technic is employed, it is doubtful that the preservation of a round pupil predisposes to the accident.

Postoperative prolapse of the iris is usually seen at the first dressing. Sometimes it is barely visible. Sometimes it is slight and is well covered with conjunctiva. Sometimes it increases in size from day to day. It may be associated with aqueous filtration.

It is not always necessary to repair a slight prolapse of the iris, and the displacement is not necessarily inconsistent with good visual acuity. In small prolapses intensive use of miotics may prevent further herniation into the wound and permit satisfactory healing. Hughes and Owens,[2] in fact, found no difference in the final visual results between cases in which small covered prolapses were let alone and those in which various surgical procedures were carried out, such as complete excision, closure with a conjunctival flap, and closure with a corneal suture. Whether operation was immediate or delayed also made no difference in their results. Allen,[4] on the other hand, makes a point of delaying for a week or more in prolapse which occurs later than forty-eight hours after operation, so that the wound may be as firmly healed as possible.

Spaeth[1] advocates a single application of the actual cautery but opposes more than one application, as inviting sympathetic ophthalmia. If the first application is unsuccessful, his preference is for surgical correction of the prolapse by resection and the van Line sliding flap.

When there is a wide gaping, corneoscleral sutures are usually essential to provide adequate protection to the contents of the eye from the outer world. Allen[4] very properly suggests that the surgeon who must operate for a prolapse of the iris should spend some time thinking about what he is going to do before he does anything. The tissues, he reminds his readers, are thin, and undue haste or pressure may cause disastrous loss of vitreous. He suggests thorough anesthesia by the retrobulbar, subconjunctival and topical routes, with enough adrenalin to reduce tension and soften the eyeball (if it is not already soft) so that vitreous will not present as soon as the incision is made. Canthotomy and cyclodialysis after excision of the prolapse are added precautions. My own preference in prolapse of the iris is to take the patient back to the operating room promptly, replace the iris under pentothal anesthesia, and close the wound with another corneoscleral suture. The eyes look and function better, in my experience, when this plan is used, and the postoperative course is smoother.

Hemorrhage. Moderate bleeding at operation, which usually occurs before delivery of the lens, may come from the incision, or from a small iridodialysis. It should be checked and the anterior chamber cleared of blood before the lens is delivered. This type of hemorrhage is seldom a serious complication.

The expulsive type of bleeding, which sometimes occurs at operation, usually follows loss of vitreous. It is not necessarily associated with hypertension, which suggests that preoperative attempts to reduce a high blood pressure are uncalled for. It is, however, related to age; it is sometimes associated with choroidal arteriosclerosis, and it may follow sudden reduction of intraocular tension, particularly in glaucoma. Most surgeons feel that when it occurs, the prognosis is hopeless and the only problem is whether to do an evisceration at once or at a later time. If the anesthesia is such that the patient is oriented, it is possible to obtain his permission to proceed at once. Lindner[5] is almost alone in believing that some eyes can be saved. His method is to make two trephining holes immediately, with a 2 mm. trephine, each 10 mm. from the limbus. He uses cocaine (0.2 cc. of a 3 per cent solution) for anesthesia and also institutes intravenous calcium therapy at once.

Expulsive hemorrhage which occurs after operation begins with violent pain, followed by chills and fever. The wound ruptures

and blood pouring from it carries with it the vitreous and the retina. Impending subchoroidal hemorrhage, if it can be anticipated, can sometimes be averted by immediate posterior sclerotomy, which permits drainage of the subchoroidal and subretinal spaces, but usually the damage is done before treatment is possible.

Hemorrhage into the anterior chamber and hemorrhage through the vitreous which appear in the coloboma of an iridectomy are treated by firm pressure dressings, morphine, and hemostatics, including thromboplastin, subcutaneously or by vein. Absolute rest is essential.

Hemorrhage into the anterior chamber following operation usually absorbs fairly promptly, but the hyphemia, which develops later, for some reason does not, particularly if the patient is a diabetic. Trauma is always a precipitating factor, but the accident may occur following routine atraumatic dressing of the wound. The initiating pain may last three to six hours. In many instances blood cells are precipitated to the lower levels of the anterior chamber with the result that the iris has a brassy look, while blood cells and blood pigment lie in grooves and folds on Descemet's membrane. An important result of late hyphemia is that the fibrin present may result in an organized exudate which bridges the pupil. Treatment consists of mydriatics, dionin to promote absorption, calcium, iodides, thromboplastin, and warm compresses.

I mention with some hesitation a dehydration regimen which I have employed with considerable success in intraocular hemorrhage over a period of years. It consists of (1) the withholding of all food and fluid, (2) the intravenous infusion of 50 cc. of 50 per cent glucose, (3) the subsequent administration of white Karo syrup in fruit juice or water every three hours during the day and every four hours during the night, and (4) the twice daily administration of ascorbic acid. Although the rationale of this method would be difficult to justify, there is no doubt in my own mind that by this means I have saved the sight of more than one patient with intraocular hemorrhage.

There might also be mentioned in this connection the report by the Eye Bank for Sight Restoration Incorporated, of several cases in which vision has been improved by intraocular injection of a small amount of the patient's own spinal fluid, which, it will be remembered, has much the same chemical make-up as vitreous humor. The blood-stained vitreous is removed through a hypodermic needle of large caliber, with a hypodermic syringe used to create negative pressure.

Delayed Re-Formation of the Anterior Chamber. At the first dressing, forty-eight hours after operation, the anterior chamber is usually found re-formed and of normal depth. Sometimes, however, it is collapsed at this time, while in other cases in which it has re-formed normally, it may subsequently be lost. In still other cases it may collapse when the corneoscleral sutures are removed, or immediately after their removal.

If the sphincter is intact, re-formation of the anterior chamber which is delayed beyond the fifth or sixth day is a potentially serious complication, although Zentmayer[6] reported a case in which re-formation was delayed until the eighteenth day without bad results. This is, however, most unusual. The most constant etiologic factor in delayed re-formation and late loss of the anterior chamber is a leaking wound, with filtration of aqueous through the corneal incision. Restlessness, coughing, squeezing of the lids and similar actions are precipitating causes, as are technical errors at operation. Frequently there is direct clinical evidence of the aqueous filtra-

tion, in the form of gaping of the corneal incision, a filtering bleb of conjunctiva, or a tear in the corneal flap. The fluorescein test is frequently useful in demonstrating these findings. Three serious complications may ensue: (1) formation of peripheral synechiae at the angle of the iris, as the result of contact between the cornea and the iris, with a subsequent predisposition to secondary glaucoma; (2) epithelization of the anterior chamber, and (3) striate keratitis, which persists as long as the iris and cornea remain in contact with each other.

Every endeavor should be made to keep the pupil mobile and thus prevent peripheral anterior synechiae. Mild mydriatics are used occasionally but for the most part it is kept constricted with miotics. Air may be injected into the anterior chamber, through a slanting incision on the cornea, though this method should be used with the greatest care, as it may cause the breakdown of the healing incision; it may be wiser to cover the incision with a conjunctival flap before it is employed. Silver nitrate may be used along the edge of a leaking wound, or, if this fails, a small leak may be sealed with diathermy. If a definite fistula is found, it should be opened at once; capsule, iris, or whatever structure is incarcerated in it should be removed, and the opening should be lightly cauterized and closed with a conjunctival flap.

Detachment of the Retina and Choroid. The incidence of retinal separation after cataract extraction, according to Hughes and Owens,[2] is as frequent after the extracapsular as after the intracapsular type of extraction, which suggests that the supporting action of the posterior capsulezonule diaphragm is not as important as it is sometimes supposed to be. In their series the incidence rose in proportion to the amount of vitreous lost, which I think is the general experience. Bagley's[7] studies suggest that the loss of vitreous may initiate degenerative changes, with the formation of traction bands, which subsequently pull off the retina. My own experience proves the logic of this reasoning.

Separation of the retina is likely to develop in cases in which the postoperative course has been stormy. It is particularly likely to follow loss of vitreous. On the other hand, the patient's history and the appearance of the affected eye frequently suggest that the condition may have existed prior to operation. Delayed discovery of the accident militates against successful re-attachment. Any detachment, in fact, which has lasted longer than two months is not usually reparable. Diathermy is fairly successful in bringing about re-attachment. According to Guyton,[3] a single application is successful in about 30 per cent of all cases and two applications are successful in another 30 per cent. If, after evacuation of subretinal fluid, the retina does not fall back into position promptly, the injection of isotonic solution of sodium chloride into the vitreous or the anterior chamber may force it back into place.

Detachment of the choroid immediateiy after a cataract operation has no great clinical significance. Some ophthalmic surgeons believe that it will be found routinely if it is looked for. It may affect any portion of the retina and may be so extensive that, as Spaeth[1] describes it, examination of the fundus reveals nothing but a gun-barrel-like tube directed toward the posterior pole of the eye. Holloway and DeLong,[8] by careful correlation of ophthalmoscopic findings and microscopic sections, showed that in most cases the condition is the result of massive choroidal and subretinal edema, following capillary transudation of serum. The circumstances of its development and the slow but consistent spontaneous recovery which usually follows are in line with this conjecture.

It was formerly the practice to restrict activity in postoperative choroidal detachment, but at the present time most ophthalmic surgeons permit the usual postoperative regimen, with perhaps a little more activity than normal, and are securing as good, if not better, results.

Prolonged choroidal detachment, with the loss or flattening of the anterior chamber which always accompanies this compli-

cation, is, of course, a serious matter, since it is likely to result in the formation of anterior synechiae between the iris and cornea, frequently with subsequent secondary glaucoma. My own plan, if I am confronted with a shallow anterior chamber and if the fluorescein test reveals no leak in the wound, is to search for a choroidal detachment, which is usually found. Treatment is begun at once with eserine salicylate (0.5 per cent) three times daily. This constricts the pupil and pulls the iris root out of an already embarrassed iris angle. My experience is that re-formation of the anterior chamber and absorption of the subchoroidal fluid usually occurs. If the desired results are not secured by this method, as of course they are not in all cases, I observe the patient for a few days longer. If at the end of a week the anterior chamber is still flat, the patient is taken to the operating room, the sclera is exposed over the detachment, a Graefe knife is slipped through the sclera, care being taken to avoid the region of the vortex veins, and the wound is held open with a Worth muscle hook until most of the subchoroidal fluid escapes. The anterior chamber often re-forms before one's eyes. If it does not, air injection is carried out.

Delayed Wound Closure. Delayed or incomplete closure of the wound through which a cataract has been removed is not uncommon. Satisfactory healing should be complete by the seventh postoperative day. Delayed healing is much less frequent since the corneoscleral or some other type of suture has been used to close cataract incisions.

If healing is delayed after capsulotomy, some capsular remnants have probably been caught in the wound. Protrusion of the vitreous may also be responsible. One method of making certain that this does not occur is to sweep the iris spatula the full length of the wound just before the lids are closed. If either of these conditions is responsible, it should be corected, by cauterization, additional corneal sutures, or conjunctival flaps, according to the necessities of the individual case.

Wound Rupture. Late rupture of a corneoscleral wound, if not caused by expulsive hemorrhage, is almost always the result of trauma. Whether or not prolapse of the iris has occurred, the ruptured portion of the wound should be uncovered and the edges should be sutured to the episcleral tissues with fine silk sutures, which then are covered with a conjunctival flap. If rupture has been extensive, the wound margins should be lightly cauterized with the actual cautery before the sutures are tied. If prolapsed iris or vitreous is present, it must be cleanly resected. Failure to care for prolapse of the iris may result in high limbal staphyloma, and extensive secondary surgery may be necessary to save the eye and prevent inevitable secondary glaucoma.

Complications Arising from Removal of Sutures. Complications are not infrequent following the removal of sutures from a corneoscleral incision, though some of them can be avoided if the sutures are properly placed, that is, if long superficial bites are taken into the sclera and the corneal lips of a somewhat shelving notch.

As a rule, sutures should not be removed before the tenth day. Adequate topical anesthesia, sometimes supplemented by akinesia of the lids, may be necessary in patients who have developed photophobia and sensitivity and in whom manipulations without this precaution would be difficult or impossible. The sutures should not be grasped with forceps before they are cut. The upper lid should be lifted with the finger and the point of a sharp Stevens scissors should be introduced from below, or horizontally, into the loop of suture, which then can be cut with safety. The aid of a loupe magnifier is essential, as is good illumination. When this method is used, if the patient moves suddenly, or is uncooperative, no harm is done; the loop of suture is merely pulled away from the point of the scissors. If difficulty is encountered when an attempt is made to remove sutures at the usual time, there is no reason why their removal should not be delayed for another week or two.

Filtering Cystoid Cicatrix. Filtering cystoid cicatrix occasionally follows capsul-

otomy extractions. Examination with the slit lamp usually shows a minimal incarceration of the iris, at the mid point of the incision lying over the mid point of the coloboma. In such cases healing has occurred with the lips of the sclera slightly offset from each other. The patient is treated as if he had had an operation for filtering cicatrix and is carefully guarded against infection, to which this condition makes him peculiarly susceptible.

Conjunctivitis. Conjunctivitis and other intraocular infections are fortunately not common in modern ophthalmic surgery. One reason is that great care is taken before operation to be certain that pyogenic bacteria are not present. This precaution is essential, since the closure of the eye after operation provides a satisfactory culture tube for any retained bacteria, the growth of which is also favored by the body temperature, the lack or movement of the parts, and decreased tear secretion.

Any infection confined to the conjunctiva, if properly treated, usually clears up satisfactorily, though it may prolong convalescence for many weeks. Spread into the anterior chamber must be prevented. General measures include (1) the use of hot sterile compresses, (2) copious irrigations with warm boric acid or warm normal saline solution often enough to keep the cul-de-sac free from pus, (3) the local and systemic use of sulfamylon (1 per cent), and (4) the local and systemic use of penicillin or streptomycin or both. These agents have largely replaced those formerly employed, such as silver nitrate, argyrol, and mercury. In conjunctivitis, as in other infections following cataract extractions, it is of the greatest importance (1) that the antibiotic drug be given in sufficiently high dosages to accomplish adequate blood levels, and (2) that its administration be continued for a sufficiently long period, so that, when it is withdrawn, reactivation of the infection does not occur.

Atropine conjunctivitis, which is not peculiar to cataract extractions, is treated as it would be under other circumstances, that is, by withdrawal of the drug, the substitution of some other mydriatic, the local use of boric acid ointment, and frequent irrigations of the cul-de-sac with warm physiologic salt solution. If it is particularly desirable that atropine be continued, this is frequently possible if 1 or 2 drops of benadryl (¼ per cent) or antistine drops are added to the solution and some one of the antihistaminic drugs is given orally.

Iridocyclitis. Iridocyclitis following cataract extraction has three chief causes:

1. It may be the result of leaving lens capsule and cortex in the eye at operation. This type of infection is for all practical purposes allergic and the picture is nonspecific. If tests for sensitization prove positive, treatment consists of desensitization by a lens protein staphylococcus toxin mixture, by the method devised by Burky and Henton.[9] Removal of the lens material left *in situ* is not advised. Its absorption is preferable.

2. Iridocyclitis may follow rupture of the hyaloid membrane of the vitreous at operation or, in some cases, at a considerable length of time after operation. It is usually a late complication, after round pupil intracapsular extraction, when it may be explained by the blocking off of the pupil with a bleb of vitreous which frequently projects forward like a mushroom into the anterior chamber.

3. The third type of iridocyclitis is a low grade and extremely obstinate infection. Chemotherapy and antibiotic therapy are ineffective and the best results are secured by typhoid vaccination combined with mydriatics and hot compresses. Short wave diathermy has also been advised.

Spaeth[1] suggests that iridocyclitis is sometimes the result of activation of a latent tuberculous lesion by the trauma of operation. If such an origin is suspected, an intradermal tuberculin test should be caried out, with appropriate treatment if it is positive. Only in the occasional case does iridocyclitis go on to sympathetic ophthalmia.

Spastic Entropion. Spastic entropion occurs only when conjunctivitis, or iritis, or iridocyclitis is present. It may continue, in

spite of efforts to control it, until the etiologic factors are corrected. If the application of slightly diverging strips of adhesive to hold the lower lid downward is not successful, external angle canthotomy should be performed without delay, since the condition causes too much irritation to permit it to continue unchecked.

Purulent Endophthalmitis. Though purulent endophthalmitis may develop weeks or even months after operation, it is usually observed at the first dressing. In most violent cases the pathologic changes include edema of the lids, edema and congestion of the conjunctiva, haziness of the cornea, exudate in the anterior chamber, and blurred red reflex. There are several possible causes, such as (1) a low grade chronic dacryocystitis following obstruction of the lacrimal ducts; (2) inadequately treated chronic conjunctivitis; (3) an acute upper respiratory tract infection; (4) superficial abscesses around the corneoscleral sutures, occasionally with direct extension of infection along the suture tract into the anterior chamber; and (5) a break in sterile technique.

Prophylactic measures include (1) careful examination of the lid margins, conjunctiva and lacrimal sac before operation, to exclude chronic infection and (2) the preoperative local use of penicillin, even in grossly clean cases, at least three times in the twenty-four hours preceding operation. Naturally, if any infection is present, operation is postponed until it is cleared up by appropriate measures, which always include penicillin. Special precautions are taken at operation to ensure absolute sterility.

The treatment of a postoperative infection is chiefly the administration of penicillin in adequate intraocular levels. Mydriatics are used to keep the pupil as well dilated as possible, and foreign protein therapy is given in the form of injections of milk or of typhoid or diphtheria antitoxin. Results are none too good with this method from the standpoint of visual acuity, and enucleation or evisceration may eventually be necessary, but in the prechemotherapeutic era the outlook was practically always hopeless. Spaeth[1] believes that if the infection is limited to the anterior segment, discission or iridotomy of organized exudate may save some eyes. He has also observed an occasional recovery following spontaneous perforation of the cornea.

Corneal Changes. The importance of prolapse of the iris and vitreous is not only the low grade iritis, which usually recedes, but the slow, progressive haziness of the cornea which may follow at an interval of some months and which is frequently responsible for serious loss of vision. Other causes of persistent corneal haziness (striate keratitis), in addition to postoperative iridocyclitis, include excessive instrumentation at operation, traumatic placing of sutures, and careless wound closure. Any of these causes may be responsible for such a degree of damage to the corneal epithelium that normal regeneration is prevented and edema persists.

In Hughes and Owens',[2] experience, persistent haziness of the cornea was more frequent after intracapsular extraction with corneoscleral sutures than after extracapsular extraction without them. To reduce its incidence they suggest the use of very fine braided silk on atraumatic needles. Care should be taken not to tie the sutures too tightly if the suture tract is unusually long. The use of a surgeon's knot is also wise, but not essential.

Nutritional changes following cataract extraction are most likely to follow a previous uveitis. The cornea becomes dull and infiltrated and the blotchy deposits on the posterior surface are quite different from the punctate keratitis present in the usual iridocyclitis. The iris becomes dull, and organized exudates bridge the pupil. Frequently considerable uveal pigment is present. Cultures are consistently negative, but the treatment is the same as that employed for frank infection. Results are slow to appear.

Epithelial Downgrowth. Downgrowth of the epithelium into the anterior chamber may be the cause of intractable secondary glaucoma following cataract extraction.

The complication can be avoided by the use of corneoscleral sutures, to insure firm closure, with careful inspection of the closed wound with magnification (my personal preference is for the 2½ power telescopic loupe) after the sutures are tied and before they are cut. The long ends, which are used for retraction purposes, make possible careful inspection of the wound for tags of conjunctiva or cornea. If any are present they can be massaged out of the wound with the curved back of a small Worth muscle hook. Those longer than 1.5 mm. should be picked up and cut off. Irradiation has been employed in the management of this complication, but neither it nor any method is effective if epithelization of the anterior chamber is extensive.

Epithelial cysts of the anterior chamber are most likely to be found in cases in which there has been delayed wound closure, as the result of early invagination of epithelium. They are difficult to treat. Needle evacuation of the fluid, followed by irrigation with weak iodine solution, is sometimes successful, as is the injection of sclerosing fluids into the cavity. Irradiation has yet to be established as an effective method.

Secondary Glaucoma. In the occasional case, ocular hypertension discovered after cataract operation may be pre-existent. It is quite possible for low grades of simple, primary, noninflammatory glaucoma to exist without detection. If it is detected, it should be treated before cataract surgery is done.

Most often glaucoma discovered after a cataract operation is related to the operation. The most common cause is iridocylitis, though it may also follow (1) failure of the anterior chamber to re-form, (2) retention of capsular and cortical fragments, (3) loss of vitreous, and (4) downgrowth of epithelium into the anterior chamber.

In some cases of secondary glaucoma miotics are effective, but as a rule surgery is required. If the angle is blocked by anterior synechiae of inflammatory debris, cyclodialysis is the procedure of choice, though some surgeons prefer corneoscleral trephining. No surgery can be performed until any inflammatory reaction which may be present is well controlled. In the interim, the glaucomatous condition is treated by bed rest, sedation, miotics, and, if necessary, paracentesis of the anterior chamber.

After-Cataract. The essential factor in the formation of an after-cataract is the persistence in the pupillary aperture of sheets and strands of anterior capsule. The remnants of the cortex and of hemorrhage may also be present. Retained cortex may play some part in the development of the after-cataract, though many authorities feel that in the absence of infection, iritis is unlikely to develop unless the lens is hypermature or is otherwise degenerated.

Various prophylactic measures have been suggested, including the use of special forceps and irrigation of the aqueous chamber. Treatment consists of discission with a sharp knife needle of the Ziegler or Wheeler type, capsulotomy, iridocapsulotomy discission with a deWecker scissors, or capsulectomy. All of these corrective procedures may themselves be attended by serious complications. Discission, for instance, may be followed by secondary glaucomatous iridocyclitis, with later phthisis bulbi, and by late retinal separation, as the result of vitreous contraction; both are usually delayed complications. If synechiae have formed, capsulectomy is contraindicated, since, if they are dense or multiple, the trauma of operation may precipitate iridocyclitis or cyclitis.

THE GENERAL STATE OF THE PATIENT

Because cataracts are chiefly found in elderly persons, surgery is attended with certain risks not commonly present in younger patients submitted to operations on the eye. The strict immobility formerly required was sometimes difficult to impose and often predisposed to hypostatic pneumonia, which might have serious consequences, as well as to thrombosis and embolism. The introduction of corneoscleral sutures has made this regimen no longer necessary. A patient operated on under local anesthesia may be placed on a low back rest immediately, and all patients, if it seems desirable, can be got out of bed

within six to twenty-four hours after operation. The patient must be told, however, that he must avoid abrupt, jerky, and jarring movements.

Foci of infection everywhere in the body, including the teeth, nose and ears, chest, and prostate gland, should be identified and properly treated before operation. Although there is not complete agreement on this point, there is no doubt that iridocyclitis may be of endogenous origin, and many authorities hold that it is more frequent after operations on patients with foci of infection.

There seems no special relationship between hypertension and the development of such postoperative complications as expulsive choroidal hemorrhages, and therefore no reason to delay operation in an attempt to reduce it. Syphilis also seems to play no part in postoperative complications. Certain complications, however, are thought to be more frequent in diabetic than in nondiabetic subjects, on the general principle that diabetics are particularly susceptible to trauma. Actually, complications in diabetic subjects are prone to occur only in those on a high insulin dosage. These complications, which chiefly take the form of an increased frequency of anterior chamber hemorrhages, are readily explained by the sclerotic condition of the peripheral circulation in diabetes. Satisfactory stabilization of the diabetic state is essential both before and after operation and calls for the services of a competent internist.

Postoperative delirium or psychosis is particularly frequent in elderly subjects. It may range from the simple mental confusion of senility to hallucinatory insanity. It is essential that patients who are used to alcohol receive it regularly, to prevent withdrawal delirium. Greenwood,[10] who estimates that postoperative delirium occurs in 2 to 3 per cent of all cataract extractions, observed no increase in the frequency of this complication in the large number of insane persons upon whom he had operated. This situation he attributes to the prophylactic use of large doses of barbiturates. There is no doubt that chloral hydrate, the bromides and similar agents are effective in preventing postoperative delirium, but they are of no value when it is once established. Moreover, when such drugs are employed in elderly subjects, they should be used in small doses, and with frequent changes of position, to prevent respiratory complications.

The importance of delirium after cataract extraction is not the condition per se, but the damage which the patient may do to the operated eye in the course of it. The reassurance of an understanding member of the family or of a friend is often of far more value than any medical treatment. Incidentally, much nervousness and apprehension at operation and afterward can be prevented by explaining to the patient beforehand exactly what is to be done, what he must do to cooperate, and what can be expected in terms of improvement of vision if the operation is a success.

Postoperative precautions against the development of complications after cataract extractions are so well known that they need only be mentioned. Straining to empty the bowel or the bladder must be avoided, and the patient may usually be permitted to get out of bed to perform these functions if that proves necessary. Coughing must be prevented by sedation, since it may cause expulsive subretinal hemorrhage and similar complications. Sneezing should also be prevented and nothing should be used at the time of dressings which would be likely to cause it. Postoperative vomiting is not infrequent, even when local analgesia has been used, and methods to prevent it must be used.

SUMMARY

Certain of the most important complications which may occur in the course of cataract operations, or which may develop in the postoperative period, have been briefly discussed. Such complications are by no means infrequent and the best way to manage them is to prevent them. They are difficult to correct when once they have become established, and the development of

one complication often leads to the development of another or of several others.

REFERENCES

1. Spaeth, Edmund B.: Principles and Practice of Ophthalmic Surgery, ed. 4, Lea & Febiger, Philadelphia, 1948.
2. Hughes, William F., Jr., and Owens, W. C.: Extraction of senile cataract. A statistical comparison of various techniques and the importance of preoperative surgery, Am. J. Ophth., 28:40, 1945.
3. Guyton, Jack S.: Prevention and management of complications of cataract extraction, Am. J. Ophth., 31:1085, 1948.
4. Allen, Thomas D.: Management of ophthalmological surgical complications, Surg., Gynec. & Obst., 74:598, 1942.
5. Lindner: Cited by Spaeth[1].
6. Zentmayer: Cited by Spaeth[1].
7. Bagley, Cecil H.: Retinal detachment. A survey of the etiology and results of treatments on phakics and aphakics. Am. J. Ophth., 31:285, 1948.
8. Holloway, T. B., and DeLong, P.: Peripheral pigmentation of the cornea associated with lenticular degeneration of the pseudo-sclerosis type, Am. J. Ophth., 12:871, 1929.
9. Burkey, E. L., and Henton, H. C.: Staphylococcus toxin combined with lens extract as a desensitizing agent in individuals with a cutaneos sensitivity to lens extract, Am. J. Ophth., 19:782, 1936.
10. Greenwood, Allen: Mental disturbances following operation for cataract, J. A. M. A., 91:1713, 1928.

BLOOD TRANSFUSION IN LOUISIANA

A PROBLEM IN ADEQUATE MEDICAL CARE

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NEW ORLEANS

PREFACE

In a limited way one may define medical care in terms of diagnostic and therapeutic services personally rendered by the doctor to the patient. This is the traditional physician-patient relationship, the very essence of the healing art, and the practitioner must closely scrutinize all extraneous factors which appear to influence it.

During the present century it has been increasingly exposed to external vectors of scientific, economic and political nature. That some of these have been beneficial is apparent by the elevated standards and broadened scope of medical care in the United States. It is no less evident that others of these forces lead toward the police state via federalization of medicine, ultimately to a form of existence by directive. This is a trend which must be vigorously resisted in every possible manner.

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The individual doctors who comprise the organized medical profession of the United States must realize that adequate medical care often entails much more than the personal services of the physician or surgeon. If he is to discharge competently his professional obligations to the patient, the practitioner must utilize a number of specialized services and facilities provided by nonmedical people. An outstanding example is the modern hospital, an institution providing important adjuncts to the practice of medicine, many of which are under nonmedical administrative control. There is in the final analysis no clear demarcation between the administrative and medical aspects of a hospital, since all administrative phases influence to some extent at least the basic physician-patient relationship. Limits of lay control must be as clearly delineated as possible, and the ultimate professional authority of the doctor no less clearly recognized. These comments are by no means intended as a reflection on administrative officers, who carry a great burden of highly specialized and detailed work, and without whom the practice of medicine would be extremely burdensome to say the least.

Statistical studies frequently analyze the costs of illness into "medical care," "hospital care" and "medicines." The writer (who is certainly no statistician) believes that there can be no true distinction between medical care (doctor's fees) and hospital care, since they are intimately related to each other and to the broad concept of the public health. Hence the term "medical care" used herein is an all-inclusive one.

It behooves the practitioner to take an active interest in the economics of medical care beyond his personal financial interest. The writer holds that the organized medical profession must concern itself with the cost of illness and must in the broadest possible sense do all in its collective power to make available the very best medical care at lowest possible cost. A small but very important part of that very best is adequate blood bank service.

BLOOD TRANSFUSION THEN AND NOW

Only a few years have passed since blood transfusion was notable chiefly as a "last resort" or desperate effort. Although the idea of elective transfusion became increasingly accepted such therapy still remained a time-consuming procedure in most instances, because compatible donors had to be located, a task sometimes requiring literally days. Postponement of major surgery because of a dearth of donor blood was a not infrequent occurrence.

Such difficulties, remember, existed when we had only to consider the four major blood groups. As knowledge of the Rh complex and human isoimmunization has evolved the situation has become progressively more complicated. Without the development of blood banks and their accessory services really adequate transfusion service would be improbable, to say the least.

In Louisiana there are at present fifteen functioning blood banks distributed as follows:

Alexandria	1	(private hospital)
Baton Rouge	2	(private hospitals)
Lafayette	1	(state charity hospital)
Monroe	1	(joint project of health department and Red Cross Chapter)
New Orleans	4	(private hospitals)
	1	(state charity hospital)
	2	(federal hospitals)
Pineville	1	(state charity hospital)
Shreveport	1	(private nonprofit foundation)
Shreveport	1	(state charity hospital)

In hospitals served by efficient blood banks the doctor merely orders blood as he does any other medication, specifying only when and how much. Now a blood bank is something more than a refrigerator with two or three containers of blood sitting on a shelf. Actually it must be a medical laboratory facility equipped and staffed regularly to collect, test and classify human blood; to store it under controlled conditions in adequate amounts of each of the eight major group-Rh classifications; to issue such blood as needed at any time. A blood bank renders both diagnostic and therapeutic services.

Most of the several hundred blood banks now in existence in the United States were established by and are operated for the benefit of specific parent hospitals, each of which has been primarily concerned with its own donor procurement problems and has followed its own ideas about blood bank procedures. Recent years have seen this somewhat narrow philosophy, influenced by a number of events, develop into a better understanding of the blood bank's relationship to the public health and safety. It has become evident that adequate transfusion service in any locality is at its roots a community problem since every member of the population, regardless of sex, age, or race, is potentially concerned as donor or recipient. Catastrophes such as the Coconut Grove fire and Texas City explosion emphasize the necessity for coordination of transfusion services with general disaster preparedness plans.

Hence, a really adequate transfusion service is one which can competently supply normal demand and can rapidly and efficiently expand in time of disaster in peace or war. It is essential that the community obligations of the blood bank be recognized by the local medical profession and hospital directors, and above all by the public. People must be indoctrinated with the idea that blood cannot be replaced by money and must be made aware of the moral responsibility of the recipient or his family to see that all loans are repaid to the bank. Further, the man in the street must be impressed with the necessity of providing for blood needs in advance whenever possible.

Obviously not every community in Louisiana is sufficiently large to maintain a blood bank. Yet no person residing in the state should be denied access to such a life-saving service. To serve people living in or near the larger cities where banks are established presents no major difficulties. What is badly needed is an extension of services into smaller towns and rural areas. This calls for the development of a statewide blood bank program, and for an excellent example of such we may look at Florida,

where the Florida Association of Blood Banks, an affiliate of the American Association of Blood Banks, has an excellent service.

The Florida Association is comprised of autonomous blood banks located in several communities about the state. Through cooperative efforts the various banks have adopted standard technical and administrative methods and have provided for interchange of blood between member banks. Rural communities take active part in the program through mobile donor units sent out by nearby banks. Blood is not sold, but all private patients who receive transfusions are billed for laboratory service charges. Charity patients are served without charge.

The Association enjoys the cooperation of the State Health Department, and the Florida Highway Patrol, the latter agency providing emergency transportation of blood by means of its radio patrol cars. The endorsement and active support of the Florida Medical Association contributed greatly to the development of the service.

The existing blood banks in Louisiana provide a structure on which, under professional leadership of the Louisiana State Medical Society, a service of similar magnitude may be provided for the people of this state. An effective program will be based on:

1. Voluntary standardization of technical procedures and apparatus on the part of the blood banks now in operation.
2. Establishment of new blood banks where needed and their coordination with existing facilities.

Since blood bank administration is so intimately related to medical practice it seems logical that the Louisiana State Medical Society should assume the leadership in developing the program, and that the component Parish Societies should take similar duties locally.

FACTORS INFLUENCING BLOOD BANK ORGANIZATION

The establishment and operation of a blood bank, either as a hospital department or a central community service, requires careful study of a number of details, notably the following:

1. Total population of the community and its normal business area.
2. Ratio between white and negro races.
3. Number of hospital beds available.
4. Type and amount of surgery being done.
5. Number of transfusions being given per month.
6. Provision of location and equipping blood bank.
7. Availability of trained personnel.
8. Blood donor procurement.

As already mentioned, the mission of a blood bank is to provide compatible blood on call at any time. No bank can continue to do this unless it receives a constant inflow of replacement donors to balance withdrawals, and further, unless it has supplementary donor lists to provide additional blood when needed. The blood bank is not intended to sell blood, but to lend it. A constant turnover is essential in maintaining a balanced supply of blood. This is the fundamental problem in blood bank administration and cannot be solved by "putting on a drive for donors". Donor recruitment must be constant; it must be on a permanent basis, and this concept must be kept in the public mind at all times.

From the purely medical point of view blood donors should be selected primarily on the questions of transmissible disease and physical ability to donate. These are the only criteria used by many blood banks in the North, where no regard is made of racial origin of the blood. However, those of us who live in the South are not permitted such an easy solution of the problem, although blood purchased from northern banks is still used in a number of southern hospitals. To meet our need a statewide blood bank program must serve all elements of the population; hence, where hospital beds are provided for negroes, there must be a corollary transfusion service. We are faced with long standing social attitudes which compel the classification and separation of bank bloods on a racial basis. However ridiculous this idea may be scientifically it exists and perforce must be recognized in any plan which may be evolved. The members of the Louisiana State Medi-

cal Society should make every effort to encourage the Negro medical leaders to solve this problem by their own efforts, aided by every possible bit of assistance that we can give them in establishing blood banks for the people of their race. Such banks already exist in certain state charity hospitals.

The majority of blood bank administrators in the United States are of the opinion that the recipient of transfusions or his family bears a moral obligation to see that all blood loaned is repaid to the bank. Existing blood banks use many different ways to publicize this, including various printed forms and circulars. However, nearly all of the independent blood banks in the United States have found that the most consistently workable means of stimulating repayment of blood loans is the so-called replacement or responsibility fee, an additional amount which is added to service charges rendered pay patients, and which is refunded as soon as replacement of blood has been made. By this means the blood banks are able to replace from 80 to 95 per cent of the blood they lend. The deficit is made up by voluntary donations from special groups and by purchase of blood in some cases from professional donors. Larger communities which serve as medical centers have a chronic problem with a blood deficit, because every year many sick people from other places come to them for hospitalization, and a considerable number require blood transfusions.

Another recourse is to appeal to the public at large for voluntary donations to make up any blood deficit which occurs. Except in the event of catastrophe such as the Texas City disaster response is usually notably poor, and even in extreme emergencies the great majority of people lose interest in a few days insofar as donating blood is concerned. This was definitely true of the Texas City case.

One may choose to disregard entirely the principle of moral responsibility for repayment and consider that the recipient and his family are not obligated in any way to repay loans. In this case one can only try promiscuous appeals to the general public. In peacetime such procedure is doomed to ultimate failure because of public indifference. People soon tire of and then disregard the high pressure publicity which such a system must employ at all times if it is to produce any blood donations at all.

If a blood bank is to function efficiently it must process from 100 to 150 transfusions regularly each month. Otherwise the turnover will be insufficient to maintain a working stock. The usual experience has been that wherever a blood bank has been established the number of transfusions per month has manifested an immediate and sustained increase. Since compatible blood is easily obtainable from the bank the doctors soon begin to make more liberal use of transfusion therapy. It can be appreciated that most hospitals of less than 200 beds would find it difficult to maintain full time blood bank service.

However, it is not necessary that all of the beds in a given community be in one hospital. They can be, as they frequently are, distributed among a number of smaller institutions. In such a situation the hospitals can pool their efforts and with community support establish a central blood bank. This can be located in its own building or on the premises of one of the hospitals. It is preferable that the center occupy its own quarters, although this arrangement is initially more expensive. In the long run it would be the most desirable arrangement because of greater efficiency, and because the glory and headaches would be more equitably distributed among the various hospitals.

Many towns, even some having hospitals, are too small to maintain blood banks. Their transfusion needs can be met in a number of ways, the most effective and economical being the "walking blood bank". This is a list of physically qualified persons who have been grouped and Rh typed and have certified their willingness to serve as donors whenever called. This type of program should be a community affair. The file of donor records is kept at some central point where it is available for reference at

any hour of the night or day. A statewide program would actually provide bank blood for emergencies in the smaller towns. Mobile unit services would allow local residents to donate and establish blood bank credits for their community in advance of need.

Since a blood bank is a medical laboratory facility it should be under medical supervision of a licensed physician, preferably one especially qualified for the field. Medical technicians must be especially trained for blood bank work. An increasing number of larger blood banks over the country, through the American Association of Blood Banks, are making available courses of instruction for technicians. The importance of adequate training is emphasized by the fact that the preparation of blood for transfusion is the only clinical laboratory procedure in which a technical or clerical error can lead directly to the death of a patient.

THE AMERICAN RED CROSS AND BLOOD BANKING

At the termination of the recent war the American Red Cross proposed to establish a national peacetime blood program by organizing blood banks in numerous communities throughout the United States. The proposal was further made that such facilities would operate complete blood bank programs including donor recruitment and the collection, processing and distribution of blood. All of this service was and is to be rendered "free of charge" to the general public. Presumably, the Red Cross would only establish a bank in a community upon invitation of the local medical society, seconded by the hospital council, and it is further presumed that it would not establish a bank in any town where the service is already in operation.

The American Red Cross has persistently maintained that its blood banks would be (and in the case of those in operation, are) under "supervision" and control of local medical societies. The writer is not certain as to just what this really means. It will be recalled that in April, 1948, the House of Delegates of the Louisiana State Medical Society went on record as opposing the intrusion of the Red Cross into blood banking, considering this to be an invasion of the practice of medicine by a lay organization. The National American Red Cross has persistently tried to establish a blood bank in New Orleans and in other Louisiana communities during the past two years. One may be assured that we have not heard the last of this, and we are not likely to.

Already the American Red Cross is operating a number of regional blood banks, for example, in Rochester, New York; Boise, Idaho; Portland and Yakima, Washington; as well as in a number of other cities. The writer has been reliably informed that many of these blood banks, indeed most of them, have been unable to meet the demand for blood in their respective communities. The writer does not believe that any of these Red Cross centers have been in operation long enough for one to establish a definite opinion about them.

Presumably because of its brilliant record in wartime donor procurement for the armed services the American Red Cross expects to obtain vast quantities of blood in peacetime by promiscuous appeal to the public. As indicated previously this concept is very badly discredited by the experience of nearly every independent blood bank in the United States. The majority of blood bank administrators believe that recipients able to do so should pay the necessary blood bank service charges, and that blood bank services should be made available without cost to deserving charity patients. Blood bank service fees are no more nor less than charges for a specialized form of medical care, and this principle is in complete accord with the traditions and usage of medical practice as we have known it in this country.

Is there actually such a thing as free blood bank service for all? The American Red Cross proposes to subsidize the program, but the money necessary to finance it must come from the purse of every one who contributes to that organization. It is more than probable that such a program would tend to discourage the fulfilment of

personal obligations on the part of recipients and their families and would invite charity where it is not needed, thus giving stimulus to the "something for nothing" idea, which is much too prevalent already. The question of "free blood" for charity patients is frequently raised. The writer cannot perceive how the status of being a charity patient in any way absolves the family of said individual of its moral responsibility of repaying blood loans. This is entirely beside the question of paying for the technical services incidental to procuring, processing and administering blood. Actually all blood per se should be free, and the ideal blood bank should function only as a trust company, having nothing for sale except service. This is one of the basic aims of the American Association of Blood Banks and is a point on which it is in entire agreement with the American Red Cross. The latter organization, however, has consistently refused to countenance the replacement fee and has appeared to ignore the combined experience of several hundred independent blood banks in this matter.

There are certain phases of blood banking in which the American Red Cross is very well suited to take an active part, such as donor recruitment, transport and canteen services, publicity, and the stockpiling of emergency supplies of blood collection and administration apparatus at strategic points about the country.

When one considers the frightful quantities of blood which would appear to be needed in event of an atomic catastrophe, or even the hundreds of pints required in less spectacular disasters, one cannot escape a belief that the American Red Cross offers the best immediately available framework on which to build a nationwide blood bank program. It is an organization highly skilled in disaster relief work, and further by means of its three thousand or more local chapters it can rapidly obtain the basic standardization of technics and apparatus so essential to an efficient national service. The American Association of Blood Banks is striving for this through its educational program and bringing standardization by the voluntary cooperation of its independent member institutions.

Returning to more local affairs the writer would personally not be upset if the American Red Cross wanted to open a blood bank in New Orleans tomorrow morning, provided he had the privilege of submitting the following questions to that organization:

1. Will Red Cross guarantee 100 per cent of the blood and plasma requirements of the community without any recourse to replacement fees?
2. What percentage of blood collected locally would be diverted from the city to the plasma fractionation program?
3. Would the Red Cross blood bank demand that all donors be bled by it and that the hospitals must stop collecting blood?
4. Would the local medical society be permitted to name the medical director and other professional workers?
5. By whom would said medical director be paid?
6. Would the medical director be in full charge of the bank, or would there be a division of authority with an administrative director?
7. Would the local medical society have the privilege of discharging the medical director or any other professional worker for due cause?
8. To what extent would research on an independent basis be permitted?
9. Would the local medical society have the right to establish minimum requirements for blood donors?
10. Would the American Red Cross consider cooperating with the existing blood banks in the community in enlisting donors and providing transport and canteen services?

Let any doctor who is faced with the problem of Red Cross opening a blood bank in his community remember to ask these questions. The writer guarantees that they are quite pertinent.

THE AMERICAN ASSOCIATION OF BLOOD BANKS

For more than eight years the writer has been engaged full time in the medical specialty of blood bank administration. More recently as a member of the Executive Board of the American Association of Blood Banks he has applied that experience in assisting to formulate one of the basic policies of the Association—that blood banking is a part of the practice of medicine and as such can best be administered under responsible control of local, independent,

nonprofit institutions under the professional supervision of local medical societies.

The Association has been endorsed in principle by the Orleans Parish Medical Society, the House of Delegates of the Louisiana State Medical Society, the American Society of Clinical Pathologists, the American Medical Association, and numerous county and state medical societies throughout the country.

The American Association of Blood Banks proposes to extend blood bank service throughout the nation and to raise its standards by means of education and the voluntary cooperation of component Institutional Members. Further, it believes that there is a definite place for the American Red Cross in this field, but that it should be restricted primarily to the nonmedical phases already mentioned.

The American Association of Blood Banks believes that in its approach to the problems discussed herein it stands on the side of the organized medical profession and is ready to assist the physician in providing his patient with an essential medical service. If the Association is to succeed therein it must have the active support of every member of organized medicine.

CONCLUSION

The organized medical profession of Louisiana bears the major responsibility for the adequate medical care of over two and one-half million people. An essential part of such care is efficient blood bank and transfusion service available in both urban and rural areas. Such a service must be adequate for ordinary demands and must be able effectively to expand to meet unusual emergencies arising in peace or war. It must further be coordinated with blood bank programs in the other states in creating a nationwide service of blood preparedness for disaster.

Blood banking, while basically a medical activity, cannot be considered as the exclusive domain of the medical profession or hospitals. Blood banks and transfusion programs must be approached as community problems. Cooperation of the general public is absolutely essential and can be obtained by soliciting the active support of every type of organization in the community. Without such cooperation blood donor procurement will be insufficient to maintain efficient blood banks.

Control of medical aspects must remain in the hands of the medical profession, which is the most logical agency to provide the active leadership which must not only initiate the program in Louisiana, but must maintain its professional standards once it has been established. Herein lies a challenge to the Louisiana State Medical Society to take up this burden of leadership.

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PREPAYMENT MEDICAL CARE PLANS

Prepayment medical care plans are a substantial part of what is needed to prevent State medicine. Their rate of progress and growth should be known and appreciated by every member of organized medicine. It will be seen by further consideration of the matter that they are of immense service to the public, and of very material service to the profession itself. So far as the public is concerned, the uncertainty of a large expenditure is replaced by the certainty of a small one. This, in brief, constitutes the advantage both to the public and to the physician.

At present, more than 34,000,000 persons are enrolled in various types of prepay surgical programs, and well over 13,000,000 are enrolled in voluntary prepay plans covering medical and/or surgical care. The majority of the latter are enrolled in medically sponsored or approved plans; these plans as many physicians know, are spoken of as "The Blue Shield" plans, of which there are now 67. By way of comparison, it is of interest to note that these have all developed since 1943. While, in contrast, the Blue Cross, which started earlier, has 90 plans covering 61,000,000 persons in the field of voluntary prepayment hospitalization insurance. The Blue Shield estimates that by the end of the current year there will be 15,000,000 persons enrolled; by the middle of 1950, approximately 17,500,000. The service rendered by Blue Shield in its 67 different organizations in 1948 was startling in amount. It disbursed $62,400,000 in payment of some 1,000,000 bills to some 100,000 physicians. Approximately 80 cents of every membership dollar is being paid out to physicians for medical or surgical care.

In months past, the AMA's Council on Medical Service has assisted with the overall promotion and coordination of all voluntary medical care plans. The national service or trade organization for the individual local medically sponsored plans was incorporated in March 1946 under the name "Associated Medical Care Plans." It is under the direction of Paul R. Hawley, M. D., chief executive officer, and Frank E. Smith, as director. L. Howard Schriver, M. D., a Cincinnati surgeon, is president of the national organization.

It is anticipated that under this leadership, in the near future, there will be a national company, which will be able to do business in any part of the nation. There are some 10,000 or more firms, which are themselves national in extent, and which desire to deal with one company and not the 67 local organizations. It is felt that such a national company would fill a very necessary part of prepayment medical care, but it is not planned that it should supersede in any way the local plans now in

operation, which are inherently best to serve the local interests.

The types of policies offered by these various plans fall into three general groups: (1) The service policy. In this the physicians offer services as scheduled in the contract at a flat fee. (2) Indemnity policy, which offers payment up to a certain amount for each type of care according to schedule. (3) The combination service-indemnity policy, in which services are provided for lower income groups but which becomes limited by an indemnity schedule when the patient's income tops a certain level. In most States these various plans are subject to the insurance laws of the State. The control of the majority of these plans is in the hands of physicians whose organizations started and financed them. Approximately two-thirds of the Boards are doctors and one-third laymen. The success of these plans is in no small degree dependent upon the cooperation of the physicians, who have concern to insure that care is limited only to that which is needed by the patient. In other words, the insurance organization pays only for necessary surgery.

The advantages of voluntary prepayment medical care are those of insurance in any field. Particularly, however, in the field of medicine the advantages are briefly, as follows: The individual is assisted in budgeting for and securing protection against the expenses of catastrophic illness. The free choice of a physician is assured and mandatory. The control of the organization remains in the hands of the doctors.

The over-all cost of medical care has increased greatly. Of this, patient's fees amount to only a small part of the increase. These prepayment plans assist in taking care of a large part of the doctor's fee. Prepayment tends to reduce the size of the population which goes to make up the indigent problem.

Louisiana Physicians Service, our own prepayment medical care plan,— thanks to the diligence and intense application to duty of the officers,—is rendering valuable service. It has now been in operation three years. As all members should know, it functions separately from the Louisiana State Medical Society, but is entirely owned by the Society. Some 40,000 individuals are covered by its policies. The annual income on these policies is in excess of $325,000 a year. In its existence so far it has paid a total of 10,000 claims, the money from which went into the pockets of the individual members of the profession. Its growth and service to the profession has been most gratifying. When we reflect on the growth over the nation as a whole of prepayment for medical care, and also consider our own situation and the valuable part that Louisiana Physicians Service is playing, we should congratulate our leaders, nationally and locally.

For too long a time have the medical organizations separated themselves from the economic aspects of medical care. At long last we are discovering how best we could serve the public interests, and incidentally, that of medicine as a whole. It should be the duty of every member of organized medicine to promote and support the operation of the prepayment medical care plans.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

AMA CLINICAL SESSION

The 1950 Clinical Session of the AMA will be held in the Nation's Capital, December 6-9. This is the third annual clinical session and attention will be focused upon the diseases and conditions most frequently met by the general practioner. The program for the session includes *scientific lec-*

tures and clinical sessions on a most interesting variety of key topics by doctors who are outstanding authorities. The *scientific and technical exhibits* will dramatize developments in the progress of modern medical practice. *Actual surgical procedures* orginating in Johns Hopkins hospital will be shown by *color television.* This will be a demonstration similar to that presented in Atlantic City during the anual meeting last June which proved of such intense interest to the doctors present.

A feature of the Clinical Session will be the award to the physician who is selected as the "General Practitioner of the Year". In the past two years this award has come to be recognized as high honor and has been a definite step toward bringing the recognition that is his due to the family doctor. The procedure for selecting the physician who is to receive this high award is as follows: Each parish and district medical society is given the opportunity to nominate a doctor; the State Society, through the Executive Committee, selects a candidate from this group, awards him recognition as the outstanding general practitioner for the year from this state and then submits his name to the Board of Trustees of the American Medical Association. The Board selects three names from those submitted by the various states and from this list the House of Delegates of the AMA selects the one man designated to receive the award of general practitioner of the year. Last year the Louisiana State Medical Society selected Dr. Charles M. Horton, of Franklin, as the outstanding general practitioner of Louisiana for 1948 and after his qualifications were reviewed by the Board of Trustees of the AMA his name was included on the list of three doctors voted upon by the House of Delegates. This was considered a great honor to Dr. Horton and to Louisiana. Nominations for the 1949 award have been received by several component societies in Louisiana and the recommendations are at this time being considered by the Executive Committee.

The Secretary of the AMA, Dr. George F. Lull, states as follows in a recent communication referring to this Clinical Session: "A record attendance of the country's general practitioners is sure to impress lawmakers and public officials with the unity and purposefulness of the medical profession. Resulting national publicity is certain to impress the general public with the extent to which doctors go to get 'post-graduate courses', thus guaranteeing patients the best and most up-to-date medical care."

The highlight of the *entertainment* program will be on Wednesday evening, December 7, when there will be a broadcast of Ralph Edward's famous radio show, "This is Your Life", followed by a stage show, to which all attending the session are invited. Key figure of the broadcast will be a general practitioner whose identity will be kept secret until the broadcast begins.

It is hoped that many physicians from Louisiana will attend this Clinical Session of the AMA which, from all indications, will be an outstanding meeting. Hotel reservations are available and these may be secured by sending request to the Chairman of the Subcommittee on Hotels, American Medical Association, Hotel Reservation Bureau, Star Building, Washington 4, D. C. For convenience of the doctors a form for requesting accommodations is carried in the Journal of the AMA each week. Be sure to attend to this without further delay.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SOUTHERN PSYCHIATRIC ASSOCIATION

The Southern Psychiatric Association, with Dr. Walter Otis (New Orleans) as president, Dr. R. Burke Suitt (Durham), vice-president, and Dr. Newdigate Owensby (Atlanta), secretary, is meeting in New Orleans on November 27 and 28, 1949. Headquarters will be at the Roosevelt Hotel.

This association is a flourishing society. It is the largest, geographically speaking, of the regional psychiatric organizations in this country, and embraces exactly the same territory as the Southern Medical Association, that is, the sixteen Southern States and the District of Columbia.

The Southern Psychiatric Association was organized in Memphis, in 1935. It has 154 members, who by heritage and training, for the most part are conservative in outlook and practice. The association has been meeting yearly since its organization, with the exception of the period of the War. Since the War, annual meetings have been resumed. The Board of Regents of the organization has set down rather rigid standards for admission to fellowship. It is reported that they prefer men who are diplomates of the American Board of Neurology and Psychiatry, or men whom they feel will likely become diplomates.

At various times, programs have been given by men from other sections. On certain occasions, the presentations have covered various points in the whole field of medicine where contact with psychiatry was significant. The program this year will consist of papers given by members and by eminent guest speakers.

The association is to be congratulated on its vigor and on the service it renders to the psychiatrist of the South.

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AMERICAN COLLEGE OF SURGEONS

Nine hundred and twenty-one initiates were received into fellowship and eight honorary fellowships were conferred by the American College of Surgeons at the Convocation on Friday evening, October 21, the closing session of the thirty-fifth annual clinical congress in Chicago.

The following from Louisiana were admitted to fellowship: Dr. Alfons R. Altenberg, Monroe; Dr. Clarence E. Boyd, Shreveport; Dr. Eugene H. Countiss, New Orleans; Dr. A. Penn Crain, Jr., Shreveport; Dr. Irving J. Glassberg, New Orleans; Dr. Glen R. Johnson, Mansfield; Dr. H. Allen King, New Orleans; Dr. Wendell H. Kisner, New Orleans; Dr. V. Hugh Price, Lake Charles; Dr. Joe D. Talbot, Shreveport; Dr. George W. Vickery, New Orleans; Dr. Simon V. Ward, New Orleans; Dr. James W. Welch (deceased) Alexandria.

The American College of Surgeons has around 15,570 Fellows in the western hemisphere and in a few countries overseas. It was organized in 1913 to elevate the standards of surgery. One of its best known activities is hospital standardization; 3,150 hospitals were on the last approved list, published in January, 1949. Clinical research, particularly in cancer; approval programs for cancer clinics, cancer detection centers, medical services in industry, and graduate training in surgery; production and approval of medical motion pictures, and literary research are among other activities.

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1949 GENERAL PRACTITIONER AWARD

The Executive Committee of the State Society has selected Dr. Charles M. Horton, as the outstanding general practitioner of Louisiana for 1949. It is felt that this is an honor well deserved by Dr. Horton, who was also selected for this recognition in 1948.

It is hoped that the Board of Trustees of the American Medical Association will accept the recommendation of Dr. Horton by the Louisiana State Medical Society for national recognition and that his name will be presented to the House of Delegates of the AMA to be voted upon for the national award. Biographical sketch and other data have been forwarded to the American Medical Association with this object in view.

Congratulations Dr. Horton, and sincere wishes for the AMA award which you so much deserve!

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WORLD MEDICAL ASSOCIATION

The first general assembly of the World Medical Association met in Paris, September 1947, with forty-five national medical organizations represented by 125 delegates and Dr. L. H. Bauer, of Chicago, was elected Secretary General. This organization was effected for the purpose of replacing L'Association Professionelle Internationale des Medecins, which was organized in 1926 and

became inactive after war began. The objects of the organization, as expressed in the By-Laws, are (1) to promote closer ties among the national medical organizations and among the doctors of the world by personal contact and all other means available; (2) to maintain the honor and protect the interest of the medical profession; (3) to study and report on the professional problems which confront the medical profession in the different countries; (4) to organize an exchange of information on matters of interest to the medical profession; (5) to establish relations with, and to present the views of the medical profession to the World Health Organization and other appropriate bodies; (6) to assist all peoples of the world to attain the highest possible level of health; (7) to promote world peace.

The House of Delegates of the American Medical Association has approved this organization and the Executive Committee of the Louisiana State Medical Society feels that members of the Society should participate in the Association in order to be kept closely informed of world events in medicine and be able to assist in raising the standards of medicine and health throughout the world.

Individual membership is $10.00 a year and checks should be sent to the United States Committee, Inc., World Medical Association, 2 East 103rd Street, New York City.

FOURTH DISTRICT MEDICAL SOCIETY

The Fourth District Medical Society held its meeting at the Shreveport Charity Hospital, October 4, 1949.

A Symposium on Pediatrics was held in the afternoon. The following papers were given: 1) "Congenital Heart Disease" by O. D. Thomas, M. D., 2) "Tularemia in Children" by Harold Levy, M. D., 3) "The Use of Alidase in Pediatrics" by Charles E. Anderson, M. D., and 4) "Infantile Eczema" by R. C. Tilbury, M. D.

A round table discussion followed, and dinner was served at 7:00 p. m.

In the evening, Dr. Clarence E. Webb, Shreveport, gave a discussion on "Newer Concepts on Treatment of Upper Respiratory Infections."

The following officers were elected: J. E. Holoubek, M. D., Shreveport, President; V. M. Fletcher, M. D., Ringgold, Vice-President; O. D. Thomas, M. D., Shreveport, Secretary-Treasurer; W. C. Gray, M. D., Springhill, Delegate; J. E. Holoubek, M. D., Shreveport, Alternate Delegate.

The retiring officers were: W. C. Gray, M. D., President; R. H. Van Horn, M. D., Mansfield, Vice-President; J. E. Holoubek, M. D., Secretary.

DR. L. ROLAND YOUNG, CLINICAL DIRECTOR, EAST LOUISIANA STATE HOSPITAL, JACKSON, LOUISIANA

Dr. L. Roland Young, formerly of Covington, Louisiana, and for the past five years at Daytona Beach, Florida, has recently accepted the position of Clinical Director of the East Louisiana State Hospital, Jackson, Louisiana. While in Daytona Beach, Dr. Young practiced endocrinology as well as neurology and psychiatry. Prior to going to Daytona Beach he was for six months Senior Psychiatrist on the staff of the Taunton State Hospital, Taunton, Massachusetts.

FIRST ANNUAL FOUNDERS DAY FORUM OF THE IBERIA PARISH AND THIRD DISTRICT MEDICAL SOCIETIES

The Medical Society of the Third Congressional District presented the First of its Annual Founders Day Forums at the Frederic Hotel in New Iberia, Louisiana, on November 19, 1949. The Iberia Parish Medical Society acted as host and members of the profession were cordially invited to be their guests. An excellent scientific program was planned for the entire day. Papers were presented by men outstanding in their respective fields.

In addition to the scientific program a round table luncheon was scheduled for noon with questions and discussion.

BOOK REVIEWS

Operating Room Technique: By Edythe Louise Alexander, R. N. 2nd ed. St. Louis, The C. V. Mosby Company, 1949, pp. 765, illus. Price, $10.00.

This is a remarkable book. It should be in the library of every hospital. It should be available to any nurse who goes near an operating room. Operating room supervisors should teach their nurses from it and should themselves practice its precepts. These are the considered opinions of a reviewer who is not ordinarily given to enthusiasms.

The author of the book, who is presently the supervisor of the operating rooms of Roosevelt Hospital in New York, has had contacts with some of the ablest surgeons in the country, as the list of acknowledgments in her two prefaces shows. Although she has undoubtedly profited from the associations, this book is first of all the individual work of an intelligent, interested, experienced, efficient operating room nurse.

The contents cover practically everything that any nurse, be she beginner, scrub nurse, circulating nurse or supervisor, needs to know about an operating room and about the procedures carried out in it. The descriptions are so clear and the illustrations so profuse—there are 668 of them—that it would seem impossible for anyone who used the

book to go wrong on the care of an operating room, the set-up for an operation, her preparation as a scrub nurse, her performance during the operation, or her work as a circulating nurse.

The text is intelligent and logical. It deals with all types of surgical procedures. The space devoted to each operation includes a definition of the procedure, an explanation of its principles, and, if it is called for, a brief anatomic description, often with illustrations; a complete list, usually with illustrations, of the instruments, linen, sutures and other items required for the special procedure, together with instructions as to what is to be laid out and what is to be kept in reserve; a detailed, well illustrated description of how the patient is to be placed on the table (one of the things this particular reviewer frequently finds difficult to have accomplished to his satisfaction); a similarly careful description of how the patient is to be draped; and finally, a step-by-step description of the operation. The latter descriptions are among the most valuable features of the book, for opposite each special step is a parallel account of exactly what instruments, sutures and other equipment are needed for that step. This would seem a practically foolproof method of presentation.

The book also contains an outline of lectures on operating room technic; a set of questions to be answered on them; suggestions and forms for grading surgical nurses' work and recording their experiences; an outline of administrative methods; and descriptions of the duties of orderlies, porters, and other operating room aides.

A great many of the illustrations have (very wisely) been borrowed from well known surgical texts. There are occasional instances in which the choice might have been wiser. Although the individual ligation of the pulmonary vessels is described, tourniquet lobectomy, which is no longer generally used, is illustrated (p. 371). Similarly, the one-suture-line method of end-to-end anastomosis is illustrated (p. 481), although most surgeons now use a double line of sutures. These are, however, minor flaws in a generally excellent production.

It is easy to understand why this book, first published in 1943, is now in its second edition, after being reprinted four times in the first edition. The author has, of course, the correct concept of operating room procedure when she writes, "It must be emphasized that the surgical team functions according to previously arranged plans, which are based on fundamental principles as well as on experience." The reviewer could wish for himself and for other surgeons no better fortune than to operate in an operating room supervised and conducted by the technic described in this book and to be assisted by nurses who had been taught from it and who, preferably, had taken another look into it just before they scrubbed up or began to circulate.

FREDERICK FITZHERBERT BOYCE, M. D.

Neurology: By R. R. Grinker, M. D. and P. C. Bucy, M. D. 4th ed. rev. Springfield, Ill., C. C. Thomas, 1949, Price, $12.50.

Admitting that he no longer practiced neurology as a discrete specialty, Grinker asked Bucy, who had worked on parts of the book before, to help him revise his book. Bucy, a neurosurgeon and professor of neurology, was an outstanding selection. There is need for such a book as Grinker planned and it is indeed fortunate that Bucy agreed to resuscitate it.

This new edition has been completely revised and many of the undesirable features of the first three editions have been deleted. It has been more than "cleaning out the desk;" it has been rewritten. Its publication is marred by typographical errors which plagued the first three editions, but when one considers that this is an 1100 page book, about a difficult and controversial subject, produced by tremendous industry and scholarship, one is being something of a criticaster to pick at its small failures.

Grinker, now a practicing psychosomaticist, has included no chapter on psychiatry though he has woven well accepted psychosomatic concepts into the whole cloth, thereby adding considerable color

Bucy has contributed a valuable chapter on the psysiology of the motor system about which his own research and clinical study give him the liberty of well formed opinions. These physiologic beliefs though they are in conformity with the consensus of neurophysiologists, are not the final word; yet Bucy's consecution is such that the general reader may not be able to delineate that which is pure conjecture. This section should be bound in looseleaf.

The third edition was divided somewhat unaccountably into thirty chapters. Now, with only eighteen chapters, the fourth edition is much more orderly, requires less searching and seems to allow more comprehensive treatment of the subject. Also the former chapters on anatomy, pathology, and physiology have been incorporated piecemeal into the body of the text. This is a distinct improvement, better correlating disease with structure and function and giving a more dynamic quality to the whole subject.

Although everything Bucy says about cerebral arteriography is true, nevertheless, he minimizes its present widespread use. His emphasis of its dangers too heavily burdened the short paragraph which he devoted to this important procedure. Injections of the intracranial arterial circulation are being done daily all over the world with success and agreeably few complications.

In a similar manner he deplores the grave dangers of surgical treatment of congenital intracran-

ial aneurysms. Although it is a touchy business, nevertheless the statement needs to be further qualified. There has been a steady advance in the surgical treatment, abetted by angiograpy, and this should be more conspicuously emphasized. If he is writing for the general medical readers, he should not close their minds to a procedure which might save the life of one of their patients, if referred to a competent consultant.

As a whole it is a fine book. Medical students, general medical readers, as well as disciples of the neurologic discipline will continue to read "Grinker" and they should.

FREDERICK C. REHFELDT, M. D.

Blood Transfusion: By E. L. DeGowin, R. C. Hardin & J. B. Alsever, Philadelphia, W. B. Saunders and Co., 1949, pp. 587 illus. Price, $9.00.

The appearance of this textbook is timely as the rapid expansion of transfusion therapy, and the extensive development of blood banks have created an acute need for a unit presentation of the many and complex aspects of the clinical as well as technical problems involved in blood transfusion therapy.

The book is designed for medical personnel whose patients receive transfusions; secondly for those who supervise transfusion services; and thirdly for technicians who perform the necessary tests and procedures.

The text is well integrated and covers a complete range of clinical, immunologic, technical, and administrative subjects relating to the transfusion of whole blood and its major derivatives. Technical procedures are carefully described and well illustrated. The authors are clinicians and the clinical aspects are well presented although occasionally the urge for explicitness leads to pedantry. This is particularly apparent in the discussion of indications for therapy.

The book is, nevertheless, an invaluable outline of the development and current practice in transfusion therapy. It is interesting, well written, and comprehensive. The bibliography is well selected and useful.

PAUL T. DECAMP, M. D.,

Radiologic Exploration of The Bronchus: By S. Dirienzo, M. D., Springfield, Ill. Charles C. Thomas, 1949, pp. 332, illus. Price, $10.75.

This splendid volume fills a void in radiological knowledge and probably will remain as one of the classical works of medical literature. It should serve as a valuable aid in the accurate diagnosis and localization of pulmonary lesions.

The first three chapters are devoted to the embryology and anatomy of the bronchus. The roentgenologic appearances in the various positions are clearly described and illustrated. Chapters IV through VII are concerned with the preparation of the patient, technics of instillation of the opaque media, bronchographic characteristics of the normal bronchus, tomography and serial exploration of the bronchus. The paragraph devoted to the physiopathology of the cough is interesting and instructive. The description of the author's technic of bronchography is clear and in detail.

Chapter VIII is concerned with bronchopulmonary malformations and the remaining five chapters cover the pathologic lesions of the lung in which bronchography is of value, such as, bronchiectasis, emphysema, carcinoma, hydatid cyst and suppuration. The physiopathology and the etiological factors in bronchiectasis are clearly illustrated and described. The chapter pertaining to carcinoma of the lung should be carefully studied by every physician who is interested in the diagnosis and treatment of these lesions.

The illustrations are outstanding because of their clarity and wealth of detail. The text is concise but the reader is uncertain, at times, of the loss due to the translation from the original language. In general, the internist, surgeon and radiologist should find this work a necessary and valuable addition to their list of useful books.

J. N. ANÉ, M. D.

PUBLICATIONS RECEIVED

Charles C. Thomas, Springfield, Illinois: Urological Aspects of Spinal Cord Injuries, by George C. Prather, M. D., Edited by Michael E. DeBakey, M. D., R. Glen Spurling, M. D., and Barnes Woodhall, M. D. The Sexual Criminal, A Psychoanalytical Study, by J. Paul de River, M. D., F. A. C. S.

Harcourt, Brace & Company, New York: Life Among the Doctors, by Paul DeKruif.

W. B. Saunders Company, Philadelphia: A Textbook of Physiology, Edited by John F. Fulton, M. D., Sixteenth Edition, Illustrated. A Textbook of Surgery by American Authors, Edited by Frederick Christopher, M. D., F. A. C. S., Fifth Edition.

Froben Press, Inc., New York: Abraham Levinson Anniversary Volume, Studies in Pediatrics and Medical History, Edited by Solomon R. Kagan, M. D.

The Blakiston Company, Philadelphia: The Development of Gynaecological Surgery and Instruments, by James V. Ricci, M. D.

Paul B. Hoeber, Inc., New York: Modern Practice in Anaesthesia 1949, Edited by Frankis T. Evans, M. B., F. F. A. R. C. S., D. A.

J. B. Lippincott Company, Philadelphia: Medicine of the Year, First Issue 1949, Edited by John B. Youmans, M. D.

Oxford University Press, New York: Acute Appendicitis and Its Complications, by Frederick Fitzherbert Boyce, M. D.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

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1948-49

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Program: Mrs. Rhodes J. Spedale, 751 Eden St., Plaquemine.
Public Relations: Mrs. C. Grenes Cole, 4938 St. Charles Ave., New Orleans.
Revision of By-Laws: Mrs. Edwin Socola, 1219 Broadway, New Orleans.
Rural Health: Mrs. M. C. Wiginton, Hammond.
Year Book: Mrs. Herman Gessner, 119 Audubon Blvd., New Orleans.

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MESSAGE OF PAST PRESIDENT OF WOMAN'S AUXILIARY

The annual meeting of any organization marks the close of one year and the start of a new and challenging twelve months. As we survey the months that have passed, we feel a very deep appreciation to all members of the executive board and to each auxiliary member who have contributed generously and efficiently of their time in extending our objects and aims.

The mid-year Board meeting was well attended and successful. The reports of the Parish presidents revealed an active, alert, well-informed membership who worked faithfully to achieve our goals. Louisiana now has a paid auxiliary membership of 900 and a total membership of 906. Some dues are delinquent—May we all strive to reach 1,000 membership in 1949-50.

During 1948-49, the state president had the added responsibility of editing our newly born Auxiliary publication News and Views. This proved to

be a real research job, very enlightening, profitable, and enjoyable albeit very taxing at times.

Two definite forward steps were taken in the general business sessions at the Convention in New Orleans when the Auxiliary voted (1) to use two separate sessions for the general business of the Convention instead of the one continuous fatiguing plan of a long business session; and (2) invite the Parish President-Elect to attend the State Executive Board meetings without the privilege of a vote. This in order that they might become schooled in auxiliary work for the job ahead.

Sincere appreciation is extended the Medical Society of the State of Louisiana for its support in general and in particular to the Council of Medical Services. Dr. Freidrichs, Chairman, and Mr. Frank Lais, Jr., Executive director, for publishing and mailing our state publication—"News and Views," to the advisory council, Drs. Grey and Murphy, who gave unselfishly of their time.

Joy and deepest gratitude fill my heart—Joy for the warmth and genuine courtesies that were extended in serving you; and gratitude for the contacts and associations which inspired me to greater effort. As we salute the old and greet the new, may I say again what a pleasure and privilege it has been to work with the Auxiliary Members of Louisiana.

Sincerely,

MRS. O. B. OWENS

WOMAN'S AUXILIARY TO AMERICAN MEDICAL ASSOCIATION

1948-49

President: Mrs. David B. Allman, 104 St. Charles Place, Atlantic City, New Jersey.

President-Elect: Mrs. Arthur A. Herold, 731 Oneonta Street, Shreveport, Louisiana.

Treasurer: Mrs. George Turner, 3009 Silver Street, El Paso, Texas.

WOMAN'S AUXILIARY TO SOUTHERN MEDICAL ASSOCIATION

1948-49

President: Mrs. Joseph W. Kelso, 307 N. W. 19th St., Oklahoma City, Oklahoma.

President-Elect: Mrs. R. C. Haynes, 755 Eastwood, Marshall, Missouri.

Councilor: Mrs. George D. Feldner, 3814 Louisiana Avenue Parkway, New Orleans, Louisiana.

PROJECTS 1949-50

1. Rural Health.
2. Hygeia.
3. Prepayment medical and hospital care.
4. Legislation.
5. Public Relations.
6. Organization.
7. Medical cultural items.
8. Nurse recruitment.

ACADIA PARISH
(Unorganized)

Bailey, Mrs. John S., Church Point.
Bruner, Mrs. J. C., Rayne.
Clark, Mrs. Leon A.
Faulk, Mrs. J. W., Jr., Crowley.

ALLEN PARISH
(Unorganized)

Buck, Mrs. Gurdon, Kinder.
Hargrove, Mrs. W. Rigsby, Oakdale.
Mayes, Mrs. James W., Jr., Oakdale.
Saint, Mrs. Charles L., Elizabeth.

ASCENSION PARISH
(Unorganized)

Baker, Mrs. Forrest E., Gonzales.
Brumfield, Mrs. D. C., Darrow.
Martin, Mrs. D. T., Donaldsonville.
St. Amant, Mrs. Guy S., Gonzales.
Thibodaux, Mrs. P. T., Donaldsonville.

ASSUMPTION PARISH
(Unorganized)

Billeaudeaux, Mrs. J. W., Klotzville.
Cox, Mrs. N. A., Napoleonville.
LeBlanc, Mrs. H. A., Paincourtville.
Pugh, Mrs. T. B., Napoleonville.

AVOYELLES PARISH
(Unorganized)

Abramson, Mrs. A. M., Marksville.
Couvillion, Mrs. W. F., Marksville.
Jeansonne, Mrs. Phillip, Plaucheville.
Jones, Mrs. H. C., Bunkie.
Temple, Mrs. H. G., Bunkie.

BEAUREGARD PARISH
(Unorganized)

Allen, Mrs. James I., Merryville.
Beckcom, Mrs. Floyd N., 13 A Pine Avenue, DeRidder.
Frazar, Mrs. John D., South Division, DeRidder.
Marcello, Mrs. Luke, 306 Port Street, DeRidder.
Roberts, Mrs. Sam T., 426 N. Pine, DeRidder.
Sartor, Mrs. T. R., 201 N. Royal, DeRidder.

BIENVILLE PARISH
(Unorganized)

Donaldson, Mrs. Buck, Castor.
Eichelberger, Mrs. Wincel K., Ringgold.
Ferguson, Mrs. R. C., Arcadia.
McKay, Mrs. Jos. Wm. III, Gibsland.
Wimberly, Mrs. F. F., Ringgold.

BOSSIER PARISH
(Unorganized)

Alexander, Mrs. P. T., Plain Dealing.
Carter, Mrs. R. O., Bossier City.
Hall, Mrs. J. B., Jr., Benton.
Landry, Mrs. L. V., 317 Yarbrough, Bossier City.
Tucker, Mrs. C. M., Haughton.
Whittington, Mrs. A. C., Bossier City.

CADDO PARISH

Mrs. L. L. Davidge, President.

(Shreveport)

Abramson, Mrs. P. D., 704 McCormick St.
Addison, Mrs. W. P., Jr., 2419 Pinehurst St.
Allums, Mrs. W. B., 1128 Janther.
Anderson, Mrs. C. E., Jr., 839 McCormick.
Anderson, Mrs. J. R., 580 Ockley.

(Dixie)

Baker, L. T., Mrs.

(Shreveport)

Bean, Mrs. Dorf, 615 Oneonta.
Bender, Mrs. N. J., 1164 Janther.
Birdwell, Mrs. J. B., 566 Longleaf.
Black, Mrs. Charles, 223 Dalzell.
Boaz, Mrs. T. D., 554 Jordon.
Bodenheimer, Mrs. J. M., 815 Delaware.
Boggs, Mrs. H. W., 140 Albany.
Boone, Mrs. C. S., 225 Patton.
Boyce, Mrs. S. W., 135 Patton.
Boyd, Mrs. C. E., 515 Elmwood.
Broyles, Mrs. J. O., 1901 Centenary.
Brown, Mrs. Wallace, 301 Unadilla.
Browning, Mrs. W. H., 1717 Marshall.
Brunazzi, Mrs. R. R., 853 Dalzell.
Bryant, Mrs. E. L., 135 Taliaferro.
Butler, Mrs. W. P., 130 Patton.
Carlisle, Mrs. J. E., 358 Albany.
Clark, Mrs. A. I., 155 Dalzell.
Cobb, Mrs. Ben, 225 Kings Highway.
Cohenour, Mrs. H. L., 715 Longleaf.
Comstock, Mrs. Glen, 344 Ockley.
Crain, Mrs. A. P., 914 Oneonta.
Crain, Mrs. A. P., Jr., 836 Columbia.
Creel, Mrs. G. A., 851 Elmwood.
Crow, Mrs. R. D., 440 Albert.
Custer, Mrs. J. L., 1162 Janther.
Davidge, Mrs. L. L., 4141 Richmond.
DeLee, Mrs. R. B., 368 Albany.
DeLee, Mrs. Stuart, 3110 Creswell.
DePriest, Mrs. F. E., 847 Kirby.
Dickson, Mrs. George, 5806 Fairfield.
Dilworth, Mrs. E. E., 646 McCormick.
Drummond, Mrs. W. F., 504 Southfield.
Duncan, Mrs. Dean H., 759 Wilder.
Eddy, Mrs. J. H., Jr., 517 Ratcliff.
Edwards, Mrs. E. Clay, 815 Ratcliff.
Erickson, Mrs. C. B., 423 Herndon.
Flake, Mrs. E. B., 3429 Beverly.
Furman, Mrs. F. S., 1756 Line.
Gallager, Mrs. Henry, 189 Archer.
Galloway, Mrs. Edgar, 575 Unadilla.
Garrett, Mrs. George, 317 Ockley.
Gavin, Mrs. J. F., 466 Albany.
Gill, Mrs. S. L., 1046 Kingshighway.
Gilmer, Mrs. P. R., 851 Olive.
Glass, Mrs. T. A., 521 Delaware.
Gorton, Mrs. J. M., Lakeshore Dr., RFD 4.
Gorton, L. W., Lakeshore Dr., RFD 4.
Gowen, Mrs. C. R., 5900 Line.
Gray, Mrs. Leon F., 1901 Centenary.
Hall, Mrs. W. M., 838 McCormick.
Hargrove, Mrs. M. D., 608 Unadilla.
Harmon, Mrs. W. S., 460 Longleaf.
Hart, Mrs. W. W., 2324 Scovell.
Harwell, Mrs. W. R., 715 Elmwood.
Hawkins, Mrs. I. F., 740 McCormick.
Heard, Mrs. J. E., 512 McCormick.
Heath, Mrs. A. G., 1617 Highland.
Heidorn, Mrs. W. B., 572 Unadilla.
Hendrick, Mrs. J. V., 448 Elmwood.
Hendrick, Mrs. T. A., 134 Boulevard.
Herold, Mrs. A. A., 1166 Louisiana.
Herold, Mrs. A. A., Jr., 431 Slattery.
Hill, Mrs. W. J., Jr., 735 Erie.
Hilton, Mrs. E. T., 901 Sheridan.
Holman, Mrs. W. C., 145 Carrollton.
Holoubek, Mrs. J. E., 172 Atlantic.
Holt, Mrs. C. S., 840 Rutherford.
Ilgenfritz, Mrs. Hugh, 360 Olive.
Johns, Mrs. Barron, 2320 Forest.
Jones, Mrs. K. B., 286 Carrollton.
Jones, Mrs. O. O., 524 McCormick.
Jones, Mrs. W. G., 145 Merrick.
Kalstone, Mrs. B. M., 4461 Finley.
Kelley, Mrs. G. P., 224 Pennsylvania.
Kennedy, Mrs. D. W., 116 E. Robinson.
Kerlin, Mrs. D. L., 648 Unadilla.
Kerlin, Mrs. W. S., 830 Ontario.
Kimball, Mrs. D. C., 110 Fremont.
Knighton, Mrs. J. E., 923 Erie.
Knighton, Mrs. J. E., Jr., 4041 Baltimore.
Knoepp, Mrs. L. F., 240 Carrollton.
La Rue, Mrs. C. L., 629 Wilder.
LeDoux, Mrs. M. S., 619 Elmwood.
Levy, Mrs. Harold, 327 Ratcliff.
Lloyd, Mrs. T. P., 6603 Gilbert.
Lucas, Mrs. R. T., 535 Pierre Mont.
Mason, Mrs. L. K., 852 McCormick.
Matthews, Mrs. M. W., 655 McCormick.
Matthews, Mrs. W. R., RFD 2, Box 96.
Mays, Mrs. C. R., 844 Oneonta.
McCook, Mrs. W. W., Jr., 179 Atlantic.
McIntyre, Mrs. D. R., 434 Unadilla.
Nicholas, Mrs. A. J., 216 Pennsylvania.
Nicholas, Mrs. S. N., 422 Boulevard.
Norfleet, Mrs. W. J., 835 Prospect.
Oden, Mrs. Pope W., 236 Gladstone.
Oxford, Mrs. T. M., 581 Unadilla.
Paine, Mrs. R. A., 651 Oneonta.
Paul, Mrs. H. W., 1535 Magnolia.
Penninger, Mrs. E. L., 1164 Janther.
Picard, Mrs. M. S., 321 Atkins.
Pirkle, Mrs. L. H., 865 Margret.
Potts, Mrs. C. H., 3814 Creswell.
Pou, Mrs. J. H., 1030 E. Kingshighway.
Quinn, Mrs. H. J., 4414 Richmond.
Reed, Mrs. Carson, 335 Gladstone.
Rew, Mrs. C. E., 1337 Oakland.
Rice, Mrs. Irwin, 427 Wilder.
Rigby, Mrs. O. C., 816 Oneonta.
Riggs, Mrs. R. H., 586 Oneonta.

Riley, Mrs. G. M., 4126 Maryland.
Rougon, Mrs. I. B., 932 Oneonta.
Rutledge, Mrs. C. P., 4334 Richmond.
Sanders, Mrs. J. C., 3218 Line.
Sanders, Mrs. J. P., 449 Galdston.e
Sandidge, Mrs. W. J., 255 Ockley.
Scott, Mrs. Leroy, 4010 Maryland.
Shavin, Mrs. J. S., 3637 Youree.
Simonton, Mrs. E. C., 808 Oneonta.
Simpson, Mrs. T. R., 731 Ockley.

Baker

Singletary, Mrs. E. W.

(Shreveport)

Smith, Mrs. B. P., Jr., 1901 Centenary.
Smith, Mrs. T. J., 535 Oneonta.
Smith, Mrs. W. W., 520 Pierre Mont Road.
Stamper, Mrs. J. R., 715 Jordan.
Strain, Mrs. T. E., 1031 Erie.
Swearingen, Mrs. D. C., 4607 Gilbert.
Talbot, Mrs. J. D., 908 College.
Taylor, Mrs. Willis, 443 Elmwood.
Thomas, Mrs. A. J., 824 Dudley.
Thomas, Mrs. O. C., 745 Ratcliff.
Tilbury, Mrs. Ross, 3433 Beverly.

(Belcher)

Tooke, Mrs. T. B.

(Shreveport)

Tooke, Mrs. T. B., Jr., 551 Ratcliff.
Trifon, Mrs. H. M., 3550 Greenway.
Vaughn, Mrs. Herbert, 142 Atkins.
Webb, Mrs. C. H., 3904 Creswell.
Wenk, Mrs. E. L., 936 Ontario.
Wilkinson, Mrs. J. A., 619 McCormick.
Williams, Mrs. T. E., 518 Atkins.
Willis, Mrs. J. C., 856 Elmwood.
Willis, Mrs. J. C., Jr., 4434 Richmond.
Wilson, Mrs. R. R., 2727 W. College.
Wolfe, Mrs. S. G., 3479 Johnette.
Womack, Mrs. R. K., 5930 E. Ridge.
Worley, Mrs. P. C., 510 Linden.
Worley, Mrs. W. B., 3220 Samford.
Yearwood, Mrs. H. M., 114 Freemont.
Yearwood, Mrs. J. G., Gayle.
Youman, Mrs. J. D., 3854 Line.

CALCASIEU PARISH

Mrs. Olin Moss, President.

(Lake Charles)

Barham, Mrs. G. E., 2009 Shell Beach.

(DeQuincy)

Bishop, Mrs. L. D.

(Sulphur)

Blanchard, Mrs. N. D., 214 Cherry.

(Maplewood)

Carter, Mrs. M. J., 43 Pine Road.

(Iowa)

Clark, Mrs. A. L.

(Lake Charles)

Clement, Mrs. E. L., 910 Pujo.
Crawford, Mrs. J. A., 1015 Pujo.
DeLaureal, Mrs. T. H., 749 Kirkman.
DiGiglia, Mrs. L. L., 2623 Shell Beach.

(DeQuincy)

Douglas, Mrs. A. E.
Drez, Mrs. D. J.

(Lake Charles)

Farmer, Mrs. W. M., 2500 Helen.
Fisher, Mrs. W. G., 1606 South Ryan.

(Port Sulphur)

Foiley, Mrs. T. H.

(Lake Charles)

Garber, Mrs. J. Y., 151 Park Avenue.
Goldsmith, Mrs. Ben, 911 Seventh.
Gordon, Miss Bernice, 1144 Hodges.
Goss, Mrs. V. V., 2452 Cypress.
Grigg, Mrs. J. R., 1128 Louisiana Avenue.
Hamilton, Mrs. J. G., 627 Pine.

(Maplewood)

Hartman, Mrs. S. F., 11 Pine Road.

(Lake Charles)

Hatchette, Mrs. C. V., Contraband Place.
Hatchette, Mrs. S. F., 2424 South Ryan.
Hebert, Mrs. Louis A., 813 Pujo.

(Sulphur)

Hodge, Mrs. J. B., 401 W. Thomas.

(Lake Charles)

Holcombe, Mrs. R. G., Jr., 133 Park Avenue.
Howell, Mrs. R. P., 814 Clarence.
Hunt, Mrs. W. E., 1640 South.
Kreeger, Mrs. George, 905 Kirby.
Kushner, Mrs. Louis, 823 Kirkman.

(Vinton)

LaCour, Mrs. E. G.

(Lake Charles)

LaCour, Mrs. Fritz, 2523 Helen.
Lamanski, Mrs. I. M., 1818 South Ryan.

(Sulphur)

Lyons, Mrs. K. M., 305 Pecan.

(Lake Charles)

Magee, Mrs. Conway, 1317 Tenth Street.

(Sulphur)

Marshall, Mrs. Robert, 611 Ruth Street.

(Lake Charles)

Miller, Mrs. M. M., 822 Ninth.
Moss, Mrs. Olin, 2011 Shell Beach.

Moss, Mrs. Walter, Drew Park Drive.
McCreary, Mrs. P. L., 1615 Grove.
McKinney, Mrs. G. C., 924 Broad.

(Sulphur)

Perkins, Mrs. D. S., 517 Huntington.

(Lake Charles)

Pierson, Mrs. M. A., Oakland.
Price, Mrs. H. V., 228 Park Avenue.
Price, Mrs. S. F., 910 Eleventh.
Raggio, Mrs. F. W., Jr., 2416 Helen.
Salmon, Mrs. D. D., 1109 Twelfth.

(Sulphur)

Seale, Mrs. W. A. K., 221 W. Elizabeth.

(Lake Charles)

Snatic, Mrs. H. S., Prien Lake Road.
Sorrells, Mrs. J. E., 115 College.
Stevens, Mrs. L., 1607 W. Common.
Topp, Mrs. O. W.
Tuten, Mrs. J. D., 1825 Hodge.

(Maplewood)

Vasquez, Mrs. G., 26 Eucalyptus Road.

(Lake Charles)

Watkins, Mrs. T. H., 2030 Drew Park Drive.
White, Mrs. H. D., 1030 Eleventh.
Whitsell, Mrs. W. R., 422 Hale.
Worden, Mrs. R. W., 822 Clarence.

CALDWELL PARISH

(Unorganized)

Hines, Mrs. S. G., Grayson.
Mangham, Mrs. A. D., Clarks.
Sherman, Mrs. D. O., Columbia.
Tugwell, Mrs. O. L., Jr.

CATAHOULA PARISH

(Unorganized)

Calliham, Mrs. P. W., Harrisonburg.

CLAIBORNE PARISH

(Unorganized)

Batchelor, Mrs. J. R., Haynesville.
Bond, Mrs. E. O., Haynesville.
Deas, Mrs. T. M., Homer.
Featherston, Mrs. John W., Homer.
Forcht, Mrs. Elaine, Haynesville.
Gladney, Mrs. James F., Homer.
Gladney, Mrs. Pat, Homer.
Middleton, Mrs. E. B., Homer.
Orman, Mrs. Mary, Haynesville.
Rivenbark, Mrs. Clotielle, Haynesville.
Waller, Mrs. W. L., Haynesville.
Wilson, Mrs. J. W., Athens.
Wolff, Mrs. Mary, Haynesville.
Worley, Mrs. A. G., Haynesville.

CONCORDIA PARISH

(Unorganized)

Coleman, Mrs. Louie B., Ferriday.
Ferguson, Mrs. E. C., Clayton.
Ratcliff, Mrs. H. B., Ferriday.
White, Mrs. Thomas, Jonesville.

DeSOTO PARISH

(Unorganized)

Armistead, Mrs. C. W., Mansfield.
Dillon, Mrs. W. E., Logansport.
Garland, Mrs. W. J., Logansport.
Jarrell, Mrs. H. W., Mansfield.

EAST BATON ROUGE PARISH

Mrs. Wiley Dial, President.

(Baton Rouge)

Albritton, Mrs. S. A., 980 Government.
Alexander, Mrs. A. S., 719 Winn Avenue.
Allen, Mrs. C. E., Jr., 1006 Ingleside Drive.
Anderson, Mrs. S. J., 658 North Fifth St.
Bannerman, Mrs. Moss, 4437 Tupelo.
Beven, Mrs. J. L., 783 Lakeland Drive.
Boizelle, Mrs. Felix, 2255 Kleinert Avenue.
Bombet, Mrs. Charles, 1744 Belmont.
Cailleteau, Mrs. E. G., 6767 Goodwood Avenue.
Campanella, Mrs. Thomas, 860 France Street.
Chiarulli, Mrs. E. E., 2952 Beech.
Cook, Mrs. W. H., 111 L. S. U. Avenue.
Cosby, Mrs. O. W., 1374 Longwood Drive.
Daniel, Mrs. J. M., 757 Laurel St.
Darby, Mrs. F. U., 2232 Terrace Avenue.
DeJean, Mrs. Ferdinand, 537 Cornell Avenue.
Dial, Mrs. W. A., 1137 Park Boulevard.
Dowell, Mrs. J. W., 1235 N. 21st.
Eidson, Mrs. W. R., 137 East Boulevard.
Fourrier, Mrs. Dan, 1230 Laurel.
George, Mrs. I. L., 1101 Steele Boulevard.
Gladney, Mrs. T. Y., Oakwood Drive.
Godfrey, Mrs. J. R., 2856 Edwards Avenue.
Griffon, Mrs. J. P., 4312 Broussard.
Hargrove, Mrs. U. S., 2727 Reymond.
Hatcher, Mrs. H. C., 1512 Arlington Avenue.
Hoth, Mrs. J. O., 1178 Richland Avenue.
Irwin, Mrs. Kernan, 2151 Lakeshore.
Jolly, Mrs. H. W., Jr., 2924 Morning Glory Avenue.
Jones, Mrs. F. J., 939 Convention.
Jones, Mrs. Jack R., 6249 Seven Oaks Avenue.
Kemp, Mrs. R. C., 616 Park Boulevard.
Kern, Mrs. L. J., 4549 Westdale Drive.
Kopfler, Mrs. M. E., Jefferson Highway.

(Zachary)

Lane, Mrs. B. B.

(Baton Rouge)

LaNasa, Mrs. M. J., 508 St. Landry.
Levy, Mrs. M. L., 1429 South 24th.
Lobrano, Mrs. I. E., 355 Hearthstone Drive.
Long, Mrs. A. D., 1367 Steele Boulevard.
Lorio, Mrs. Cecil, 3131 Dalrymple Drive.
Lorio, Mrs. L. F., 1046 Drehr Avenue.
Marks, Mrs. Paul, 2135 Parker Drive.
McConnell, Mrs. C. S., 3100 North Boulevard.
McGehee, Mrs. J. W., Jr., 339 Oakwood Drive.
McGehee, Mrs. J. W., Sr., 2174 Wisteria.

McHugh, Mrs. Jeff, 2335 Oleander.
McMahon, Mrs. Rhett, 210 Convention.
Miller, Mrs. D. S., 2521 Rhododendron.
Morris, Mrs. C. T., 2258 Terrace.

(Zachary)

Morris, Mrs. H. S.

(Baton Rouge)

Mosely, Mrs. Charles, 4121 North Boulevard.
Mullins, Mrs. J. H., 624 Park Boulevard.
Nelken, Mrs. B. E., 1416 Perkins Road.
Phillips, Mrs. J. R., 701 Winn Avenue.
Pickell, Mrs. F. W., 2177 Oleander.
Powers, Mrs. J. R., 803 Park Boulevard.
Rabalais, Mrs. R. Roy, 2143 Myrtledale.
Riche, Mrs. H. Guy, Jr., 2220 Myrtle.
Sabatier, Mrs. J. A., 4362 Sweetbrier.
Selser, Mrs. R. E., 1957 Cloverdale.
Silvey, Mrs. M., 1563 Perkins.
Simmons, Mrs. McHugh, 510 East Boulevard.
Singletary, Mrs. W. B., 613 Drehr Avenue.
Smith, Mrs. C. P., 500 Dubois.
Stander, Mrs. Alvin, 2616 Perkins.
Stephenson, Mrs. R. T., 2123 Perkins.
Toups, Mrs. J. E., 1956 Cloverdale.
Van Gelder, Mrs. D. W., 2220 Government.
Vennard, Mrs. W. O., 2476 July.
Voss, Mrs. C. H., 2125 Kleinert.
Walker, Mrs. M. A., 1269 Longwood.
Wallace, Mrs. Robert, 2285 Kleinert.
Williams, Mrs. Lester, 739 Convention.
Witter, Mrs. H. B., 2335 Myrtle.
Young, Mrs. R. W., 228 Kernan.

(West Baton Rouge)

Landess, Mrs. D. W., Port Allen.
Landry, Mrs. Paul B., Port Allen.
Thomas, Mrs. George E., Port Allen.

EAST CARROLL PARISH
(Unorganized)

Cain, Mrs. Frank A., Lake Providence.
Davis, Mrs. J. P., Lake Providence.

EAST FELICIANA PARISH
(Unorganized)

Jackson, Mrs. Paul T., Clinton.
Nolan, Mrs. Leonard S., Jackson.
Roberts, Mrs. Wm. J., Jackson.
Toler, Mrs. E. M., Clinton.
Wilkinson, Mrs. Wm. E., Jackson.

EVANGELINE PARISH
(Unorganized)

Aswell, Mrs. Charles J., Jr., Ville Platte.
Dupre, Mrs. R. E., Ville Platte.
Freeman, Mrs. O. L., Basile.
Savoy, Mrs. Frank, Mamou.
Vidrine, Mrs. Arthur, Ville Platte.

FRANKLIN PARISH
(Unorganized)

Bostick, Mrs. John N., Gilbert.
Jones, Mrs. Henry E., Wisner.
Rogers, Mrs. J. D., Winnsboro.

GRANT PARISH
(Unorganized)

Brian, Mrs. N. M., Sr., Montgomery.
Donaldson, Mrs. D. V., Dry Prong.

IBERIA PARISH
Mrs. Edwin Landry, President.

(New Iberia)

Bourgeois, Mrs. D. E., 439 E. St. Peter.
Brown, Mrs. C. P., Main Street.
Carstens, Mrs. Walter, 615 Center.
Crawford, Mrs. Lewis, Marie Street.
Dalton, Mrs. Orien, Jeanerette Road.
Dauterive, Mrs. Henry, 424 E. Main.
DeGravelles, Mrs. C. C., 635 Marie.
Dupuy, Mrs. Jules, Marie Street.
Flory, Mrs. H. M., 717 Julia.
Gajan, Mrs. I. W., Jr., P. O. Box 145.
Gonzales-Romo, Mrs. Baltazar, 406 S. Pellerin.
Landry, Mrs. E. L., 601 Marie.
Landry, Mrs. E. N., 625 Main.
LeBourgeois, Mrs. Paul A., 640 E. Main.
Pharr, Mrs. J. N., Gorham Place.
Slipakoff, Mrs. Leon, 523 Iberia.
Tolson, Mrs. John, Olivier Building.

(Jeanerette)

Villien, Mrs. L. M., 1519 W. Church.

IBERVILLE PARISH
Mrs. R. D. Martinez, President.

Eby, Mrs. Cyril, Labauve Avenue, Plaquemine.
Levy, Mrs. Simon C., 700 Labauve Avenue, Plaquemine.
Holloway, Mrs. Eugene, 809 Baist, Plaquemine.
Martinez, Mrs. R. D., 605 Court, Plaquemine.
Melton, Mrs. Edward C., 321 Haase, Plaquemine.
Miller, Mrs. Maxwell, 807 Bayou Road, Plaquemine.
Musso, Mrs. J. P., North Main Street, White Castle.
Spedale, Mrs. Rhodes, 701 Eden Street, Plaquemine.
Tomeny, Mrs. Frank, South Main Street, White Castle.

JACKSON PARISH
(Unorganized)

Blume, Mrs. Ernest, Jonesboro.
Green, Mrs. Leonard, Ansley.
Robinson, Mrs. George H., Hodge.

JEFFERSON DAVIS PARISH
Mrs. R. S. Kramer, President.

Arceneaux, Mrs. Rosemond R., Welsh.
Brunt, Mrs. Charles D., Jennings.
Harrell, Mrs. Forrest Worth, Jennings.
Kramer, Mrs. Richard S., Jennings.
Martin, Mrs. Claude A., Welsh.
McClure, Mrs. John G., Welsh.
Miller, Mrs. R. Frank, Jennings.
Romero, Mrs. John, Welsh.
Sabatier, Mrs. Harold, Elton.

Shirley, Mrs. Louis E., Jennings.
Shirley, Mrs. L. E., Jr., Jennings.
Smith, Mrs. Morgan, Jennings.
Taylor, Mrs. Kendrick, Jennings.
Turner, Mrs. Dan B., Jennings.

LAFAYETTE PARISH
Mrs. J. Boring Montgomery, President.

(Lafayette)

Azar, Mrs. P. J., 314 W. St. Mary Blvd.
Bourgeois, Mrs. Ralph, 504 S. Buchanan.
Burdin, Mrs. J. J., 431 Buchanan.
Daly, Mrs. O. P., 1316 Lafayette St.
Davis, Mrs. F. H., 832 St. John.
DeMier, Mrs. A., 314 W. St. Mary Boulevard.
Duhon, Mrs. J. O., 730 Jefferson.
Fernandez, Mrs. J. M., 311 W. St., Mary Boulevard.
Gardiner, Mrs. George V., 504 S. Buchanan.
Harrell, Mrs. Edward M., 840 S. Washington.
Kurzweg, Mrs. Paul, 312 W. St. Mary Boulevard.
Long, Mrs. L. B., 431 S. Buchanan.
Miles, Mrs. John, 314 W. St. Mary Boulevard.
Montgomery, Mrs. J. B., 518 Oak Avenue.
Olivier, Mrs. C. K., 406 Oak Avenue.

(Scott)

Prejean, Mrs. L. A.

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Kleinpeter, Mrs. E. A., Thibodaux.
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Glass, Mrs. J. B., Natchitoches.
Moreland, Mrs. W. E., Powhaton.
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Andrews, Mrs. John D., 1643 State St.
Ane, Mrs. Joseph N., 3221 State St. Drive.
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Barkoff, Mrs. Sam, 8320 Palmetto St.
Barnes, Mrs. G. C., 2407 General Taylor St.
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Bernhard, Mrs. Robert, 3427 S. Broad St.
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Bethea, Mrs. Oscar, 1453 State St.
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Blackshear, Mrs. S. M., 623 Bourbon St.
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Blum, Mrs. Henry N., 1837 State St.
Boles, Mrs. James H., 716 Broadway.
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Boyer, Mrs. Richard C., 4975 Miles Drive.
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Caire, Mrs. Arthur III, 2130 Octavia St.
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Kaplan, Mrs. Murrel, 4601 S. Claiborne St.
Kelleher, Mrs. Robert C., 1462 State St.
Kirkpatrick, Mrs. Milton E., 1426 St. Andrew St.
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Marino, Mrs. Frank X., 8432 Palmetto St.

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Phillips, Mrs. Percy A., 3113 La. Ave. Parkway.

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Rein, Mrs. W. J., 3613 Napoleon Ave.
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Roeling, Mrs. William, 4665 Franklin Ave.
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Rosenthal, Mrs. Simon, 2632 Napoleon Ave.
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Schneider, Mrs. George T., 49 Audubon Blvd.
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Scott, Mrs. Jos. T., Jr., 3429 Octavia St.
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Seeman, Mrs. William, 1577 Henry Clay Ave.
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Simon, Mrs. Sidney K., 465 Audubon Blvd.
Simon, Mrs. Theodore, 1300 Third St.
Smith, Mrs. Earl Conway, 3426 Upperline St.
Smith, Mrs. George L., 1308 Broadway.
Smith, Mrs. Victor C., 2016 Calhoun St.
Smyth, Mrs. John, 7 Richmond Place.
Socola, Mrs. Edwin, 1219 Broadway.
Sodeman, Mrs. W. A., 2037 General Taylor St.
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Souchon, Mrs. Edmond, 3126 Octavia St.
Souchon, Mrs. Marion, 3530 Octavia St.
Spizer, Mrs. David, 4705 Mandeville St.
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Stern, Mrs. Sol, 624 Broadway.
St. Martin, Mrs. Maurice, 1920 Audubon St.
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Taquino, Mrs. George, 18 Fontainebleau Drive.
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Tedesco, Mrs. Victor, 3732 Canal St.
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Thomas, Mrs. H. Ashton, 5968 Canal Blvd.
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Vickery, Mrs. George, 4922 Baronne St.
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Weed, Mrs. John P., 816 Burdette St.
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Coon, Mrs. Henson S.
Cummins, Mrs. J. W., 3910 Spurgeon.
Cutler, Mrs. Hayden, 624 Loop Road.
Flinn, Mrs. C. B., 2309 Myrtle.

(West Monroe)

Gallaspy, Mrs. G. T., 209 North Seventh.

(Monroe)

Gray, Mrs. C. P., 115 Louisville.
Gray, Mrs. C. P., Jr., 1200 Fairview.
Guerriero, Mrs. H. E., 504 Speed.
Hamilton, Mrs. A. S., 1300 Island Drive.
Hammonds, Mrs. F. H.
Hill, Mrs. C. H., 1617 Park Avenue.
Hirsch, Mrs. D. I., 301 Hudson Land.
Hunter, Mrs. M. W., Loop Road.
Johnson, Mrs. C. U., 504 Forsythe.
Kelly, Mrs. J. D.
Klam, Mrs. N.
LaCour, Mrs. B. J., 607 Glenmar.
Lobrano, Mrs. C. M., 2808 Gordon.
Marx, Mrs. F. A., 2213 Jasmine.
McConnell, Mrs. J. E., 1509 North Fifth.
McHenry, Mrs. A. G., 1810 Riverside.
McHenry, Mrs. M. A., 1000 Jackson.
Milam, Mrs. D. T., 1704 Island Drive.
Miller, Mrs. A. V., 211 Telemaque.
Mintz, Mrs. Stanley.
Moore, Mrs. D. M., 1209 Park Avenue.
Murphey, Mrs. J. W., 1012 Jackson.
Perot, Mrs. P. L., 1405 Park Avenue.
Peters, Mrs. A. L., Edgewaters Gardens.
Pracher, Mrs. John, 1403 Emerson.
Rizzo, Mrs. F. P., 1111 North Fourth.
Sampognaro, Mrs. V. J., 3402 DeSiard.
Schonlau, Mrs. J. W., 1800 North Fifth.
Shlenker, Mrs. L. L., 504 Hilton.
Simonton, Mrs. R. M., 702 Hilton.
Snelling, Mrs. J. G., 2101 Marie.

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Spencer, Mrs. B. E.

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Spencer, Mrs. W. L.
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Talbot, Mrs. Ralph, 200 Arkansas.
Tisdale, Mrs. A. D., 2924 Gordon.
Varino, Mrs. G. A., 211 Pine.
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Webster, Mrs. W. H., 1700 North Second.
Wilds, Mrs. C. E., Jr., 112 Glenmar.
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Wright, Mrs. G. W., 1005 S. Grand.
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(Pineville)

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(Alexandria)

Barker, Mrs. H. O., Horseshoe Drive.

(Lecompte)

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(Alexandria)

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Calhoun, Mrs. S. L., 2241 Thornton Court.
Cappel, Mrs. J. T., 1741 Thornton Court.
Chicola, Mrs. V. F., 1710 Stanford.

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Fendler, Mrs. Ben, 2117 Marye.
Forsyth, Mrs. H. P., 2010 Jackson.
Foster, Mrs. M. H., 2107 Albert.

(Pineville)

Foster, Mrs. R. H., Central Louisiana Hospital Grounds.

(Alexandria)

Freedman, Mrs. R. J.

(Pineville)

Freeman, Mrs. M. S., Central Louisiana Hospital Grounds.

(Alexandria)

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Gahagan, Mrs. H. Q.
Gancy, Mrs. E. R., 1937 Jackson.
Gremillion, Mrs. L. D., 2038 Albert.

(Lecompte)

Hair, Mrs. M. J.

(Alexandria)

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Hensel, Mrs. A. E.
Herrington, Mrs. C. P., 2510 Avenue A.
Honigman, Mrs. M., 2006 Turner.
Jones, Mrs. Earl, 631 Jackson.
Kingsley, Mrs. D. M., 2779 Hill.
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Landrum, Mrs. J. H., 2203 White.

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Packer, Mrs. J. M., 1776 Jackson.
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Pearce, Mrs. M. B., 1001 City Park Boulevard.
Pierson, Mrs. Clarence, Mimosa Place.
Pincus, Mrs. E. R., 2206 Jackson.
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Rand, Mrs. P. K., 2736 Georges Land.

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Richardson, Mrs. O. J., 208 Fairmount.

(Alexandria)

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Salmon, Mrs. D. D.

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Smith, Mrs. G. W., 1936 Thornton Court.
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Uhrich, Mrs. E. C., 2327 Vance.
Wallace, Mrs. R. B., Jr., 607 20th Street.

(Pineville)

Welch, Mrs. James, Central Hospital Grounds.

(Alexandria)

Welch, Mrs. J. W., 529 Park Place Drive.
White, Mrs. H. A., 2613 Vance.
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Willis, Mrs. J. T.
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Ellender, Mrs. S. Ernest, Crescent Boulevard, Houma.
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Byrd, Mrs. E. H., 1305 Texas St., Leesville.
Johnson, Mrs. W. M., 804 Dennis Ave., Leesville.
Reid, Mrs. W. E., 800 West Third, Leesville.
Shaw, Mrs. Edgar Molloy, 1501 Texas St., Leesville.
Talbot, Mrs. Milton Wm., 1001 Fifth St., Leesville.
Younger, Mrs. J. B., Younger Hospital, Kurthwood.

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Pepe, Mrs. John L., Bogalusa.
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Garrett, Mrs. J. M., Cotton Valley.
Garrett, Mrs. W. R., Springhill.
Gray, Mrs. W. C., Springhill.
Mims, Mrs. D. D., Minden.
Richardson, Mrs. S. M., Jr., Minden.
Sentell, Mrs. C. S., Minden.
Tompkins, Mrs. R. C., Minden.

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(Unorganized)

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Everett, Mrs. William, Oak Grove.
Jarrell, Mrs. C. M., Epps.

WEST FELICIANA PARISH
(Unorganized)

Niebergall, Mrs. Mary R., St. Francisville.

WINN PARISH
(Unorganized)

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Martin, Mrs. Roy V., Winnfield.
Mosley, Mrs. John T., Winnfield.
Watson, Mrs. Arthur, Winnfield.
Wright, Mrs. Roy Wm., Winnfield.

SECOND DISTRICT

Mrs. P. A. Donaldson, President.

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(New Orleans)
Cashio, Mrs. Paul, 4429 Jefferson Highway.
Casteix, Mrs. M., Box 193, Kenner.
Clayton, Mrs. J. E., Norco.
Donaldson, Mrs. P. A., Reserve.
Fernandez, Mrs. J. R., Wallace.
Gelbke, Mrs. C. F., Gretna.
Gidman, Mrs. P. T., Harvey.
Godchaux, Mrs. P. M., Reserve.
Gregoratti, Mrs. J. V., Gretna.
Gros, Mrs. Remy, Laplace.
Johnson, Mrs. A. Z., 418 Metairie Road, New Orleans.
Johnson, Mrs. Paul, Lutcher.
Kirn, Mrs. J. D., Luling.
Kopfler, Mrs. J. S., Kenner.
Labruyere, Mrs. P. P., 4480 Fourth, Marrero.
Landry, Mrs. P. T., Norco.
Massony, Mrs. A. A., Westwego.
Massony, Mrs. J. J., Westwego.
Nobile, Mrs. A. J., Lutcher.
Pavy, Mrs. A. B., Lutcher.
Ray, Mrs. Thiesen A., Harahan.
Riera, Mrs. R. B., Marrero.
St. Amant, Mrs. Alvin, Reserve.
Sellman, Mrs. A. H., 2526 Metairie Road, New Orleans.
Trigg, Mrs. Dan, Reserve.
Troxler, Mrs. F. A., Lafitte.
Wild, Mrs. Fred, Destrehan.

WOMAN'S AUXILIARY TO THE LOUISIANA ACADEMY OF GENERAL PRACTICE

This is a new venture in the auxiliary life of Louisiana and is intended to bring about closer social ties with the wives of doctors who practice in this field of medicine. As the roster is reviewed it will be noted that most of the members are active in the parish, state and national auxiliaries, many of them rendering valuable service. They are welcomed as a new group and are extended good wishes for a high degree of success.

WOMAN'S AUXILIARY TO THE LOUISIANA ACADEMY OF GENERAL PRACTICE

Mrs. Dorman B. Barber, President.
Pineville, Louisiana.

FIRST DISTRICT:

Councilors:
Mrs. N. J. Chetta, New Orleans, Louisiana.
Mrs. Bruno F. Mancuso, New Orleans, Louisiana.

Blandino, Mrs. V. P., 5400 Gen. Diaz St., New Orleans.
Burger, Mrs. O. J., 2036 Palmer Ave., New Orleans.
Cabibi, Mrs. C. P., 3027 Paris Ave., New Orleans.
Cabiran, Mrs. Louis R., 5902 Dauphine St., New Orleans.
Chetta, Mrs. N. J., 70 Fontainebleau Dr., New Orleans.
Ciolino, Mrs. Jos., 4486 St. Roch St., New Orleans.
Danton, Mrs. B. F., Jr., 2301 Tennessee St., New Orleans.
Fatter, Mrs. Esmond A., 3421 Canal St., New Orleans.
Feldner, Mrs. George D., 3814 Louisiana Ave. Pky., New Orleans.
Furlow, Mrs. T. E., Jr., 6210 Franklin Ave., New Orleans.
Gehbauer, Mrs. Louis J., 2723 Odin St., New Orleans.
Gillaspie, Mrs. R. E., 2722 Broadway, New Orleans.
Gillaspie, Mrs. W. S., 35 Versailles Blvd., New Orleans.

Guidry, Mrs. Edwin R., 720 Broadway, New Orleans.
Hyman, Mrs. David, 4012 Vendome Place, New Orleans.
Johnson, Mrs. A. E., 3130 Elysian Fields, New Orleans.
Jung, Mrs. T. A., 1219 Rendon, New Orleans.
Kirn, Mrs. Theo F., 4744 Franklin Ave., New Orleans.
LaNasa, Mrs. Philip P., 2425 Esplanade Ave., New Orleans.
Mancuso, Mrs. B. F., 2109 Alvar St., New Orleans.
Medina, Mrs. I., 2 Virginia Court, New Orleans.
Menendez, Mrs. J. C., 4210 S. Broad St., New Orleans.
Murphy, Mrs. D. J., 127 S. Solomon St., New Orleans.
Parker, Dr. Alma M., 2701 Jefferson Ave., New Orleans.
Pitkin, Mrs. A. B., 3439 Prytania St., New Orleans.
Rateau, Mrs. J. B., 6015 St. Charles Ave., New Orleans.
Robinson, Mrs. C. R., 407 Marine Bldg., New Orleans.
St. Romain, Mrs. M. J., Jr., 3831 Frenchmen St., New Orleans.
Saleeby, Mrs. Philip J., Box 6029, New Orleans.
Strange, Mrs. W. R., 5719 Clara St., New Orleans.
Tardo, Mrs. C. J., 2209 Broadway, New Orleans.
Trist, Mrs. N. P., 948 Friscoville, Arabi.
Wheeler, Mrs. C. J., 3800 Piedmont Dr., New Orleans.
White, Mrs. J. A., Jr., 30 Farnham Place, Metairie.

SECOND DISTRICT:

Councilor:

Mrs. Earle J. Clayton, Norco, Louisiana,

Atkinson, Mrs. J. W., 1009 Amelia Ave., Gretna.
Clayton, Mrs. J. Earle, Norco.
Donaldson, Mrs. Pierre A., Reserve.
Gauthier, Mrs. W. K., 208 Sycamore Dr., Metairie.
Gelbke, Mrs. C. F., 8 Willow Dr., Gretna.
Gray, Mrs. J. B., 6020 St. Charles Ave., New Orleans.
Hindelang, Mrs. F. M., 905 Monroe, Gretna.
Kopfler, Mrs. J. S., Kenner, Louisiana.
LaBruyere, Mrs. P. P., Marrero.
Massony, Mrs. J. J., Westwego.
Wild, Mrs. F. A., Jr., Destrehan.

THIRD DISTRICT:

Councilor:

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Boudreaux, Mrs. L. M., Erath.
Bourgeois, Mrs. R. L., 518 Brooks St., Lafayette.
Dalton, Mrs. O. E., New Iberia.
Davis, Mrs. F. H., 403 Myrtle Place, Lafayette.
Duhon, Mrs. J. O., 211 Cherry St., Lafayette.
Flory, Mrs. H. M., New Iberia.
Gonzales-Romo, Mrs. B., Jeanerette.
Hamilton, Mrs. Chas. E., 1124 St. John St., Lafayette.
Horton, Mrs. Chas. M., Franklin, Louisiana.
Jones, Mrs. G. R., Lockport.
Kurzweg, Mrs. P. H., Jr., Lafayette.
Melancon, Mrs. William, Carencro.
Prejean, Mrs. L. A., Scott.
Robichaux, Mrs. P. A., Raceland.
Robichaux, Mrs. R. E., Raceland.
Sonnier, Mrs. Wm., Jr., Franklin.
Voohries, Mrs. H. C., Jr., 312 Dunreath St., Lafayette.

FOURTH DISTRICT:

Councilor:

Mrs. J. M. Bodenheimer, Shreveport.

Bodenheimer, Mrs. J. M., 815 Delaware, Shreveport.
Clark, Mrs. A. I., 614 Med. Arts Bldg., Shreveport.
Cohenour, Mrs. H. L., 715 Longleaf Dr., Shreveport.
Cook, Mrs. Byron L., Minden.
Curtis, Mrs. H. P., Mansfield.
Gill, Mrs. S. L., 1046 Kingshighway, Shreveport.
Hill, Mrs. W. J., Jr., 725 Erie St., Shreveport.
Hilton, Mrs. E. T., 104 Physicians & Surgeons Bldg., Shreveport.
Huckabay, Mrs. L. S., Coushatta.
Landry, Mrs. L. V., 113 Bobbie St., Bossier City.
Pou, Mrs. J. G., 1030 Kingshighway, Shreveport.
Richardson, Mrs. T. A., Minden.
Sanders, Mrs. J. P., 3218 Line Ave., Shreveport.

FIFTH DISTRICT:

Councilor:

Mrs. John N. Bostick, Gilbert.

Allen, Mrs. Dean H., Tallulah.
Bostick, Mrs. John N., Gilbert.
Carrierre, Mrs. S., Jr., Winnsboro.
Carroll, Mrs. E. L., Columbia.
Mangham, Mrs. A. D., Clarks.
Rogers, Mrs. Hollis T., Winnsboro.
Teer, Mrs. Lorenz, Delhi.
Terral, Mrs. Forrest M., Lake Providence.
White, Mrs. Thomas B., Jonesville.

SIXTH DISTRICT:

Councilor:

Mrs. Thos. Y. Gladney, Baton Rouge, La.

Baker, Mrs. F. E., Gonzales.
Cali, Mrs. S. J., 600 W. Robert St., Hammond.
DeLeo, Mrs. S. J., Independence.
Feder, Mrs. A. J., Hammond.
Fourrier, Mrs. D. J., 1230 Laurel St., Baton Rouge.
Genovese, Mrs. C. R., Independence.
Gladney, Mrs. T. Y., 2885 Eugene St., Baton Rouge.
Lane, Mrs. B. B., Box 176, Zachary.
Levy, Mrs. M. L., 1429 S. 24th St., Baton Rouge.
Lipscomb, Mrs. C. P., Ponchatoula.
Lubritz, Mrs. E., Amite.
McConnell, Mrs. C. S., 3100 North Blvd., Baton Rouge.
McGehee, Mrs. W. R., Franklinton.
Martin, Mrs. J. D., Jr., 2926 Perkins Rd., Baton Rouge.

Morris, Mrs. Harry S., Zachary.
Pepe, Mrs. John L., 611 S. Columbia St., Bogalusa.
Pittman, Mrs. M. L., 301½ Boston St., Covington.
Rabalais, Mrs. Roy R., 2143 Myrtledale.
Simmons, Mrs. W. M., 510 East Blvd., Baton Rouge.
Wiginton, Mrs. Marquis C., Hammond.

SEVENTH DISTRICT:

Councilor:

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Bishop, Mrs. Louis D., DeQuincy.
Brooks, Mrs. Wm. F., Vinton.
Douglass, Mrs. A. E., DeQuincy.
Gray, Mrs. Lofton F., Oakdale.
Hargrove, Mrs. M. V., Sr., Oakdale.
Hargrove, Mrs. W. Rigsby, Oakdale.
Harrell, Mrs. F. W., 1019 Cary Ave., Jennings.
Hodge, Mrs. J. B., Jr., Sulphur.
Mayes, Mrs. J. W., Oakdale.
Price, Mrs. Steve F., 910 11th St., Lake Charles.
Romero, Mrs. John R., Box 564, Welsh.
Seale, Mrs. W. A. K., 401 S. Irwin St., Sulphur.
Shirley, Mrs. Louis E., 919 N. Cuttings, Jennings.

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Councilor:

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Barber, Mrs. D. B., Pineville.
Blake, Mrs. B. C., Lecompte.
Brian, Mrs. N. M., Jr., Alexandria.
Forsyth, Mrs. H. P., Alexandria.
Fraser, Mrs. S. F., Many.
Herrington, Mrs. C. P., Alexandria.
Trax, Mrs. David L., Jena.
Weber, Mrs. Gus F., Many.
White, Mrs. James A., Jr., 1740 Jackson St., Alexandria.

WOMAN'S AUXILIARY TO THE LOUISIANA ACADEMY OF GENERAL PRACTICE

While it is a matter of commonplace knowledge that after the eventful first step, the human infant can be relied upon to put the other foot forward with increasing regularity, the similarity does not extend to infant organizations. The Woman's Auxiliary to the Louisiana Academy of General Practice extended itself beyond its own wildest dreams at the first annual convention which made history in women's organizational circles. Now, due to reorganizational plans on a national scale, the group is faced with its second convention plans in a half year span.

December 11 and 12 have been set for the dates of the Alexandria meeting. Each auxiliary member is not only urged to accompany her husband, but to persuade at least one member of her professional community to qualify as a member so that his wife may also participate in auxiliary activities.

Since the auxiliary was founded on a keynote of purely social objectives, it has already become the custom for fast friendships to be formed while committee members are planning festivities. Our husbands have long had the opportunity of meeting kindred souls who share their problems and frustrations. We women are discovering compensations and privileges in our treasured positions as wives of general practitioners.

Active Mrs. Barber, president of the Louisiana group of women (the foremost in the nation) found time to attend a meeting of the sixth district group at a center just outside of Hammond. She outlined the efforts the Louisiana women had made to establish a national auxiliary at this time, and explained why a negative vote of the nation's board of the American Academy of General Practice, then meeting in Kansas City, did not mean that a national group was being frowned upon. It has been decided that it was better to have the majority of the states organize women's groups first, so that national ideals and activities may become more crystallized.

Mrs. Barber is calling a meeting of her board coincidentally with the board meeting of the men's group on November 6 at Alexandria. It is to be hoped that she will have a full attendance to help her plan her schedule for the coming year, and approve the convention committee's engagements as lined up for December 11 and 12.

Sixth district is to be heartily commended, not only for the excellency of its program on September 28, but for the interest which drew members from as far away as Alexandria, Shreveport and New Orleans. In addition to the scientific program of the afternoon, Dean John Francis McCloskey of the school of pharmacy of Loyola University, spoke to the doctor-wife dinner group on a subject of mutual interest, "Government Controls", with emphasis on the proposed Truman program of federalized medicine.

After dinner the groups diverged into meetings of separate concerns, and eleven o'clock found the far-from-home loath to leave.

If for nothing else, Hammond will long be remembered as the home of the first president of the first Woman's Auxiliary to a group of the American Academy of General Practice, and it was entirely fitting that this group be entertained by Mrs. Wiginton at her lovely home.

During the afternoon, there was informal discussion in relation to the St. Louis meeting in February. We are even expecting to charter space on several pullmans for the Louisiana group.

Come join us some time. Any member is welcome at any district meeting, and all districts are enjoined to notify the state publicity chairman of impending meetings or past news of interest.

Mazie Adkins Guidry,
Chairman of Publicity.